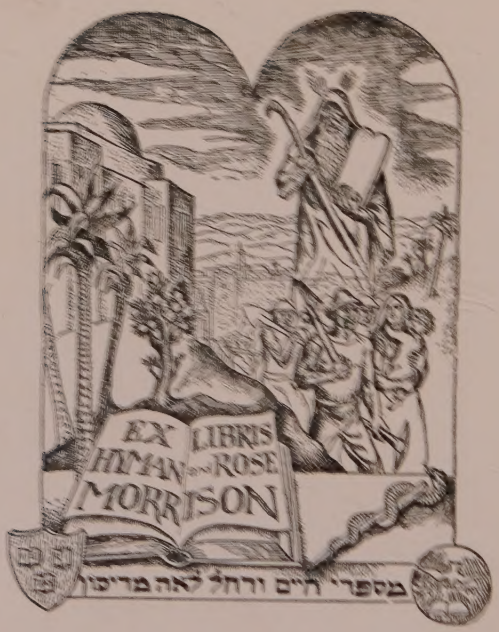
APPLIED ELECTROCARDIOGRAPHY

PARSONNET AND HYMAN



To

Dr. Hyman Morrison

with grateful appreciation

and best wishes

Albert S. Hyman

1939

APPLIED ELECTROCARDIOGRAPHY

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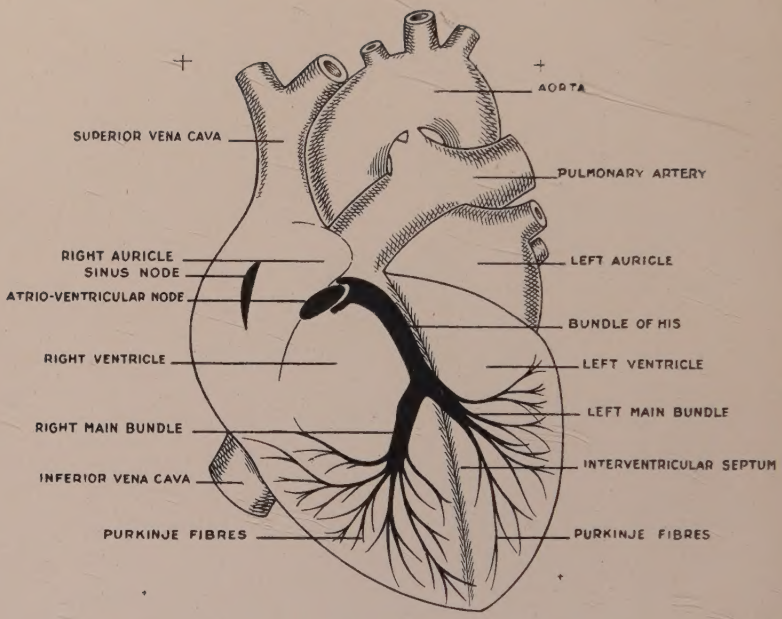

SCHEMATIC DRAWING OF HEART SHOWING CONDUCTING SYSTEM

"PHANTOM HEART."

APPLIED ELECTROCARDIOGRAPHY

An Introduction to Electrocardiography for Physicians and Students

BY

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WITH A FOREWORD BY

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TO OUR PARENTS

THIS BOOK

IS AFFECTIONATELY DEDICATED

FOREWORD

Electrocardiography has come to stay. Up to date its most valuable contributions have been to our knowledge of the physiology of the heart mechanism. In the future its chief value should be to clinical medicine in its explanation of the pathology of the diseased heart.

The technique of electrocardiography has up to date occupied much of the literature devoted to the subject of electrocardiography. This is a subject of which the clinician need have but a general knowledge for the reason that to be valuable, the work must be done by technicians entirely familiar with the instrument and with the manner of its application. This demands a special training and knowledge which should not be attempted by the clinician, but with the interpretation of the graph and its application to cases of circulatory diseases, the practicing physician must be capable of exercising his own knowledge and of determining his own interpretation of the clinical features of the case, with which he should be more familiar than the specialist or mere technician.

The peculiar value of this present volume is in that it is designed for the education of the practitioner in this art, written as the book is by clinicians, rather than physiologists or technicians.

Couched in direct terms, designed to fit the practitioner for the understanding of the information which the electrocardiograph may give him in the understanding and management of his own cases of circulatory disease, this book fulfills a real need.

HARLOW BROOKS.

PREFACE

Greater strides have been made in Medicine during the last fifty years than in the period from the almost mythical Galenus to the present time. Thus, the physician making an honest effort to keep up with the rapid progress of the medical sciences, is almost staggered by the difficulties and mass of detail encountered in textbooks devoted to these advances, and after severe trial to his time and patience, gleans only a few data of real practical value to his profession. Too many books are written for the specialist, and too few for the great mass of good general practitioners.

In the instance of electrocardiography, this is more than true. Since the monumental work of Waller and Einthoven, on the graphic interpretation of the heart beat through the aid of electrocardiography, volumes have been written on the subject, practically none of which is simple enough for the uninitiated to understand. Simplicity and clarification are the keynote of our book; we shall attempt to explain the mechanism of the heart impulses and the interpretation of the electrocardiogram, and if the reader will bear with us, we trust he will be rewarded with a clear understanding of the graphic records, which, after all, are not so complicated.

We can not leave this page without acknowledging our gratitude to Dr. Harlow Brooks, whose rare experience, constructive criticism, and helpful advice have served us well in the preparation of this volume.

To Mr. J. N. Myers and Mr. W. C. Hall of The Macmillan Company our most sincere appreciation for their

patience and cooperation is herewith expressed, and for the efforts of Mr. Samuel G. Ziger in faithfully executing the line drawings in this book our thanks are given.

Aaron E. Parsonnet
Albert S. Hyman

INTRODUCTION

Probably no mechanism of the entire mammalian make-up has aroused the speculative interest of the physiologists and anatomists more than that of the heart. From the days of the pioneer scientists who noted first with wonder and then with admiration the regular movements of an exposed human heart, to the classic experimental work of Wenckebach and Winterberg, the remarkable series of events that take place whenever a cardiac cycle is produced have never ceased to amaze and fascinate the medical student. Generations of philosophic, speculative, and finally scientific literature have appeared upon this organ. With each succeeding generation there has come out some new device or method whereby greater knowledge of the heart's action could be obtained by the practicing clinician.

The first mention in the literature in regard to the clinical study of the heart and pulse is many centuries old; Auenbrugger, however, in a remarkable monograph published in 1760, pointed out for the first time the principles involved for the percussion of the chest, and so laid the foundation for modern clinical methods. Laennec's discovery that the heart sounds could be heard more clearly through a rolled tube of paper was made in 1816. Stephen Hales brought to the clinician the phenomenon of blood-pressure, this reaching a point of perfection with the work of the great physiologist, Ludwig. Potain, in 1867, made the first satisfactory graphic records of the heart's activity. Prior to this, in 1856, Koelliker and Mueller first demonstrated the presence of a current of action in the heart. Waller in 1887, through the use of his capillary electrometer, first demonstrated the possibility of registering the

human heart beat, but his method proved to be impractical, and the first satisfactory curves from the mammalian heart were obtained by Bayliss and Starling in 1892. Roentgen made actual visualization of the heart possible through the use of his X-ray. Finally, in 1903 Einthoven introduced the string galvanometer, and this principle is used in modern electrocardiography.

Thus have appeared one by one the stethoscope, blood-pressure apparatus, the art of percussion, the polygraph, and lastly the electrocardiograph; all serving to present to the physician new and better information in regard to the functional integrity of the cardiovascular system.

So detailed and so complex has become the information about the heart, derived from these various methods of examination, that the physician to-day stands confused and perplexed when confronted with the problem of examining the heart at the bedside. From this short historical synopsis the reader can readily appreciate that the venturesome medical man of any period has been confounded by perpetually changing theories and discoveries; no doubt the future holds no sinecure for the oncoming generation of the scientifically inclined.

No one can leave the experimental physiological laboratory without being carried away by a feeling almost akin to reverence for the remarkable mechanism that regulates the orderly beating of the heart. So impressed have been many observers that the thought of a perfectly functioning engine is brought to mind, in which the opening and closing of valves, the contraction of chambers, and the regulation of volume output, all serve to complete the illusion.

Carried away by their enthusiasm, many investigators have pronounced the heart as the most perfectly functioning organ of the entire human mechanism, and much has been written and discussed in regard to the perfection of this structure. It is, therefore, almost heresy to point out that while it is true that the cardiovascular system, as it is

found in the mammal to-day, unquestionably represents the highest efficiency of functional economy, at the same time, from the point of view of pure physical science, the heart presents certain incomplete structures which in health have no significance, but which in disease are responsible for the enhancing of certain pathological changes. For example: pure science would have constructed a four-chambered pumping system with six valves instead of four. If there were a valve at the sinus venosus at the entrance of the right auricle, and if there were a similar valve at the entrance of the pulmonary vein to the left auricle, the sequelæ which follow the stenosing of the mitral or tricuspid valves would not happen. Nature, however, has not been incognisant of the need of such valves, because the organism readily appreciates that contraction of the right or left auricles forces the blood in two directions; namely, through the respective auriculo-ventricular valves as well as backward through the approaching veins. Certain ill-defined bands are found in the right auricle, while the pulmonic veins also have a series of rudimentary shelves which tend to act as baffle plates in preventing the blood from back-flow into the lungs. Thus, a purely mechanical criticism of the heart as a pumping machine shows it to be not absolutely efficient.

If fault may be found with the contracting and valvular system itself, more is it true of the specific mechanism which controls the technical elements of the pump, and as we will have occasion to point out in detail in subsequent pages, many of the irregularities of the heart beat are directly enhanced by the imperfect distribution of the stimulus for muscular control.

Reverting again to the simile of the combustion engine with its valves and contracting chambers, the specific system of the heart is compared to the electrical system of the motor, where electrical energy is manufactured from a generator and is distributed by wires to the respective firing

cylinders. The difference, however, between the firing system of a gasoline engine and the heart is that in the former the electrical impulse is carried without interruption to its end point, whereas in the heart the impulse having been generated, must find its way through the auricles without the assistance of a definite pathway, and finally striking the lower node, is carried now to its terminal point. What happens between the time that the impulse leaves its birthplace at the pacemaker and the time it reaches the auriculo-ventricular node is unimportant in the normal heart, but in the cardiovascular system already altered by disease, this deficiency in the mechanism assumes a sinister aspect.

Concerning the mechanism of the heart beat, many long and complete treatises had been written. The names of Rothberger, Vaquez, Bainbridge, Sherrington, Lewis, Starling, Eppinger, Winterberg, Mackenzie, Wenckebach, and many others, are indelibly impressed upon the literature of this study. For those, however, who have neither the time nor scientific make-up needed for a thorough perusal and contemplation of such works, the attempt is made in this volume to reduce the knowledge amassed by pioneer investigators in this field to simple truths, essential in the everyday experience of the practicing physician.

No apology is therefore made in attempting to present problems of the utmost scientific abstractness in terms of bedside simplicity. Neither do we apologize for the omission of discussions of highly problematical and debatable hypotheses. We will, however, make free use of the established phraseology of the cardiological laboratory, feeling that the terms employed to describe electrocardiographic findings will soon become an integral and familiar part of the physician's vocabulary.

We recognize full well the apprehensive attitude displayed by most physicians at the mere mention of the word

ELECTROCARDIOGRAM, and it is the hope of the authors that this volume may dispel that "inferiority complex" that surrounds the doctor when he contemplates the use of the electrocardiograph.

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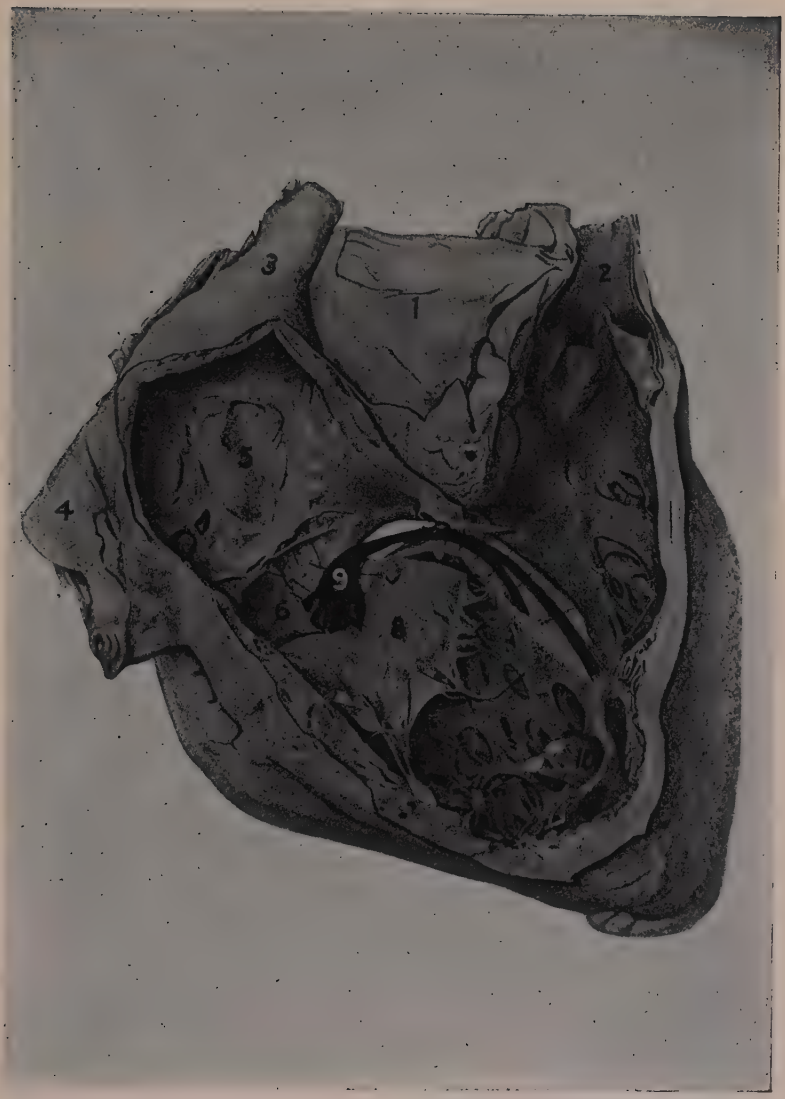


Fig. 1.—Anatomical dissection of the heart (after Tandler) showing the conducting system as viewed from the right side. The following areas are marked: 1. Aorta, 2. Pulmonic Artery, 3. Superior Vena Cava, 4. Inferior Vena Cava, 5. Interauricular septum, 6. Sinus Coronarius, 7. Filament from Node of Tawara, 8. Tricuspid Leaflet, 9. Auriculo-ventricular Node, 10. Anterior Papillary Muscle. (From Wenckebach and Winterberg.)

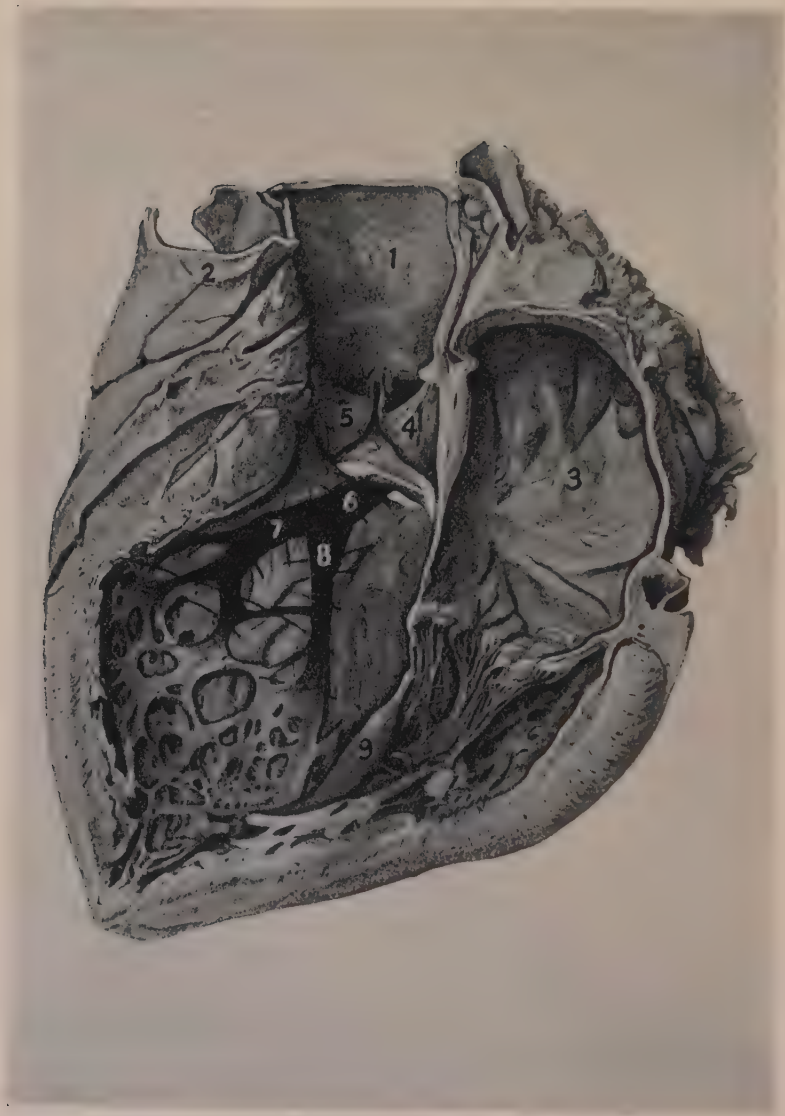


Fig. 2.—The same preparation viewed from the left, showing: 1. Aorta, 2. Pulmonary Artery, 3. Left Auricle, 4. and 5. Aortic Cusps, 6. Left Main Branch of the Bundle of His, 7. and 8. Anterior and Posterior Divisions. 9. Papillary Muscle.

CHAPTER I

PHYSIOLOGICAL BASIS OF ELECTROCARDIOGRAPHY

THE PHYSIOLOGICAL BASIS OF ELECTRO-CARDIOGRAPHY

Considerable experimental evidence has demonstrated that every muscle contraction is associated with certain electrodynamic phenomena. That part of a muscle which is contracting becomes electro-negative to the inactive portion of the same muscle segment. Further investigation has shown that this electrodynamic change occurs just prior to the actual mechanical contraction of the muscle fibre; this difference of electric potential is called by physiologists *the action current of muscles.*

The heart muscle exhibits this same phenomenon, but as the action currents are very weak, special instruments are required to demonstrate and measure this electrical energy. Such an instrument is known as the *electrocardiograph,* and the records produced by such an instrument are known as *electrocardiographic tracings,* or *electrocardiograms.*

In common with all other types of muscle, but exhibiting with particular specialization the various myogenic properties, the heart muscle is an interesting example of coördinated functional integrity. While every portion of the heart muscle retains some elements of its embryological functions: irritability, conductivity, contractility, stimulus production, and tone, because of its coördinated function, various parts of the heart have developed one or another of these properties to the exclusion of all others. Thus we find that stimulus production and irritability have been assumed by the sinus and auriculo-ventricular nodes, conductivity by the bundle system of His, contractility by the ventricular walls, and tone by the tissues surrounding the valve orifices.

Under normal conditions, these specialized portions of the heart maintain an orderly mechanism and there is no

interference or competition among the various parts for supremacy. When, however, such normal relations are disturbed by disease or unusual conditions, each part of the heart may now assume functions not usually within its sphere, yet granted to it by a common embryological origin. Thus, we may find that stimulus production or irritability instead of being developed by the nodal tissues now arises from contracting portions of the heart muscle. In other words, the pacemaker may suddenly move from the sinus node to the left ventricle.

In the normally functioning heart the mechanism of the cardiac cycle has been carefully studied. It has been found that the pacemaker of the heart lies in a small group of cells situated in the posterior wall of the right auricle near the opening of the superior vena cava at an area known to the anatomists as the *sinus venosus*. This group or "nest" of specialized cells seems to combine the characteristics of both cardiac muscle and nerve tissue, and occupies a space about 2 cm. long and a few mm. in diameter.

A secondary or lower group of specialized cells is found at the auriculo-ventricular junction; histologically, it resembles the upper node, except that it is continuous with a well defined bundle of fibres which runs downward in the interventricular septum. At the level of the papillary muscle insertions of the tricuspid valve, this bundle of fibres divides into two groups, a left and right branch, each of which continues downward in the wall of the respective ventricles and finally terminates in a very complicated network lining the two ventricles and known as the *Purkinje system*.

The stimulus, having been generated at the sinus node, sweeps through the auricular muscle and reaches the lower or auriculo-ventricular node. From this point the impulse gathers speed and rapidly passes down the bundle tissue to the two main branches and from there to the Purkinje system.

In the exposed mammalian heart it can be shown that if

an electrode be placed over the sinus node and one over the apex of the heart, the sinus node shows electro-negativity first. If this small current is recorded by a suitable photographic instrument, a tracing showing three major waves will be noted.

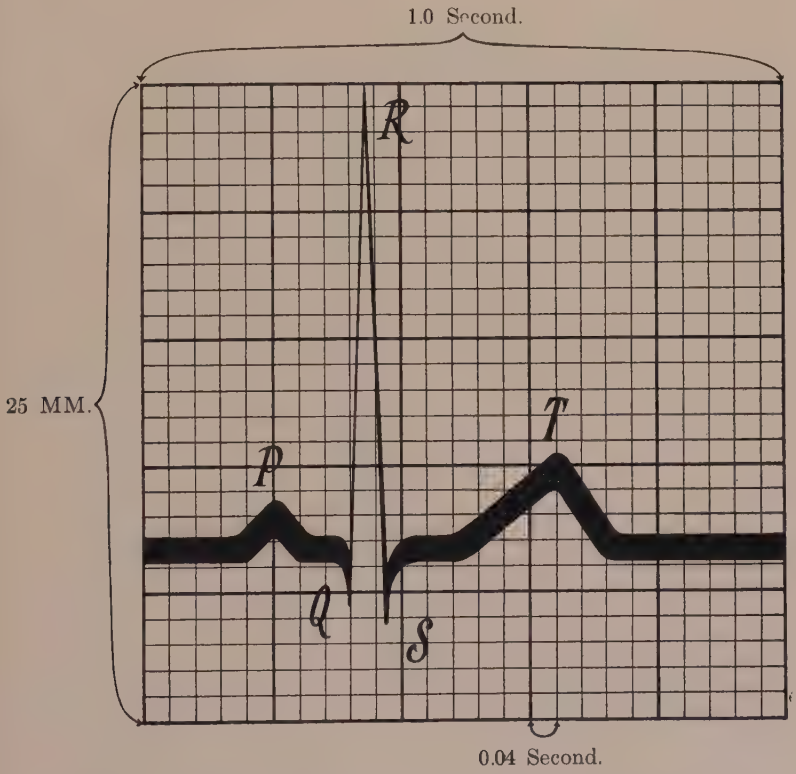


Fig. 3.—Waves seen in normal record. Schematic drawing.

These three waves have retained their original physiological nomenclature and are known as the P, R, and T waves. These names need not be confusing and have no special significance; they are kept because of their universal acceptance, and only serve to recall the work of Waller, Ludwig, and others who were pioneers in this field. Many

of the waves which were originally described and which were blessed with alphabetical names have in the years which have followed, been found to be artifacts or unimportant, and one by one they have been disregarded, until now only six waves are recognized, and three of these, the Q, S, and U waves are relegated to unusual conditions.

The P wave has long been identified with contraction of the auricles; it is normally well formed and upright. It may, however, be very small and sometimes inverted or downward. In general, the P wave can readily be identified in that it immediately precedes the largest excursions of the entire cardiac cycle. The QRS complex, known also as the *initial ventricular complex* or R wave, is the most striking element in the entire tracing because of its size.

The time relations of the various portions of the cardiac cycle are of considerable importance both as to diagnosis and prognosis. Normally, it has been found that the time interval from the beginning of the P wave to the upstroke of the QRS complex is remarkably constant, measuring about 0.16 seconds in nearly all mammals. This constant figure, however, must be modified to include time intervals measuring as high as 0.20 seconds, and as low as 0.14 seconds. Any time intervals above and below these limits are generally regarded as being indications of altered auricular physiology.

The second wave, as has been mentioned above, and the most conspicuous, is the QRS complex. This complex has been identified with contraction of the ventricles, and experimental evidence has associated it especially with the muscular elements of the ventricular chambers. The complex consists normally of a large, sharp pointed, and upright wave, suggestive of a spear point or church steeple. Original descriptions of this wave included two small waves immediately preceding and following it. These small waves are directed downward, and according to the old nomenclature, were known as Q and S waves. Their presence has been

retained in the phrase—"QRS complex," but concerning their individual significance, recent investigations have shown them to be relatively unimportant. In a presentation such as this, we may dismiss them with a simple acknowledgment of their existence, and the reader, if interested further, will find considerable academic speculation in regard to the Q and S waves in the larger specialized monographs on this subject.

The QRS complex or the R wave is ordinarily the most simple and easily recognizable wave in the electrocardiographic tracing, and the beginner will do well to identify these waves first. As has been already noted, it is usually upright in all leads. Its base occupies a time interval measuring .06 seconds in all mammalian hearts. It is never less than this, but its lengthening beyond 0.10 seconds, is ordinarily considered as evidence of myocardial change.

The third wave of the electrocardiographic sequence is known as the T wave, which to-day occupies a stage of excited controversy. Many conflicting theories are current in explaining the significance of the T wave itself, and a bitter controversy exists in regard to its alteration. So far as the beginner is concerned, the T wave, which is also known as the *terminal ventricular complex,* is associated with the vascular elements of the ventricular chambers, in contrast to the initial ventricular complex which has been identified with the muscular elements of the ventricles. Experimental work has clearly indicated that deliberate interference with the coronary circulation by ligation of one of the main branches, changes the form of the T wave. Pathologically, T wave alteration is found in any condition which interferes with the coronary circulation, such as thrombosis and occlusion.

The T wave follows the QRS complex; it is an upright rounded or blunted wave, which stands in sharp contrast to the steep sharp-pointed R wave. Normally, it is found upright in all leads, but it may be found flattened, absent,

or even inverted in the third lead. By the older physiologists such T wave changes in the third lead were considered normal variations, but there is some reason to believe that certain significance can be put on such deviations.

The physiological cardiac cycle divided into its presystolic, systolic, and diastolic phases, is readily identified in the electrocardiogram, where the P-R interval indicates the presystolic period, the QRST group the systolic period, and the T-P interval the diastolic pause.

Electrodynamics of the Cardiac Mechanism.

As has been indicated before, the electrocardiogram is merely a photographic record of changes registered by a complicated instrument which is able to isolate the very small electric currents developed by the heart. These electric currents are of minute magnitude, and the greatest of these, are measured in terms of thousandths of volts. The millivolt is the standard unit of measure for such differences of electrical potential.

In animal experimental work the obtaining of these small electrical currents is a simple procedure, as suitable electrodes may be placed directly on exposed surfaces of the heart. It has been found that the two electrodes necessary to make up any completed electric circuit can be placed at any two points on the heart, and tracings can always be obtained. Subsequent investigations demonstrated that inasmuch as the heart occupied a triangular area within the thorax, the base of the triangle being upward and the apex downward, the greatest amount of energy could be obtained by placing the electrodes at the angles of this triangle. This made possible three points at which electrical circuits could be made; one, which included a point at the extreme right of the base of the triangle and extreme left, another, which included the extreme right and the apex of the triangle, and, finally, one which included the extreme left and the apex. It was soon discovered that normally each

of these three circuits gave similar electrocardiographic records, but that in small alterations from the normal, and especially in diseased conditions, great deviations could be noted in comparing the three circuits.

In the course of time, these three circuits became known as LEADS; the *first lead* being the circuit including the extreme right and left points of the base of the cardiac triangle, the *second lead,* the extreme right point and the apex of the triangle, and finally, the *third lead,* the extreme left point and the apex of the triangle. To utilize this extremely valuable information revealed by the exposed heart in the human subject, now became the object of careful inquiry by investigators. Waller, in 1888, demonstrated that it was not necessary to apply the electrodes directly to the exposed heart, but that the electrical energy developed by the heart was transmitted to all portions of the body. Altogether he was able to demonstrate more than sixteen possible places where the electrical impulses could be satisfactorily recorded; these included both hands, both feet, the nose, ears, mouth, rectum, and vagina. In his epoch-making monograph, published in 1888, Waller showed that for all practical purposes these various points of contact could be limited to three:—the two arms and left leg.

Applying the nomenclature already developed by the physiological laboratory, Lead one was found to be best obtained from the right and left arms; Lead two, from the right arm and left leg; and Lead three, from the left arm and left leg.

In thus simplifying the electrodynamic physiology of the heart, we have intentionally omitted some considerations of fundamental importance. The electrical energy of the heart, its transmission, and its ultimate distribution have been studied in great detail and with such accuracy, that mathematical formulae have been developed. These formulae have indicated that the heart not only anatomically occupies a diagonal position within the thoracic cavity,

but that the actual electrical axis also assumes a diagonal direction. The various electric functions derived from the formulae can be simply demonstrated by reference to the diagram below.

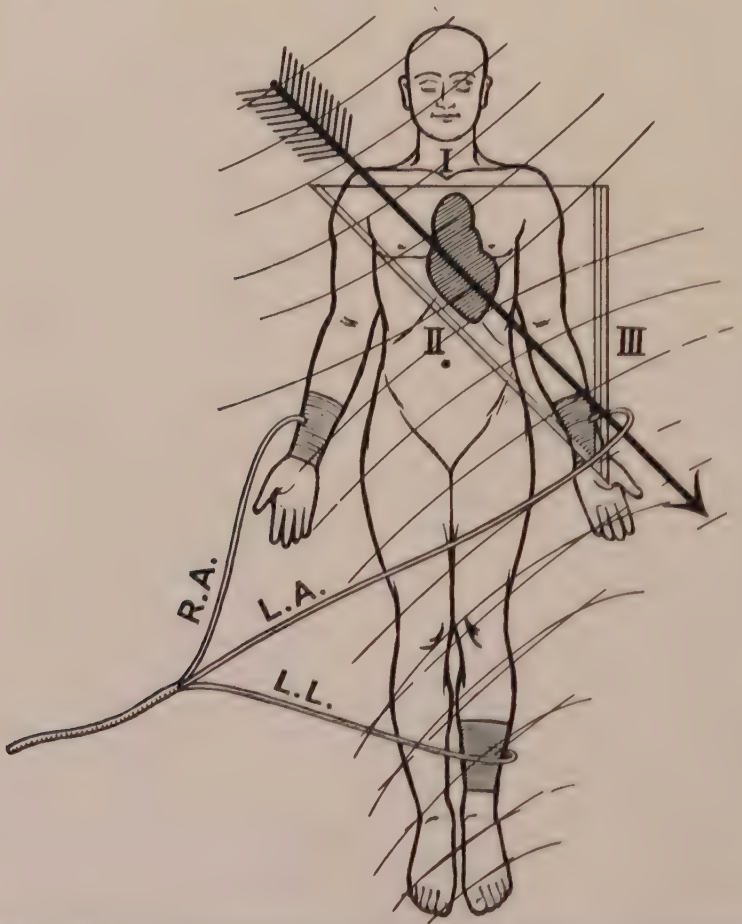


FIG. 4.—The electrocardiographic triangle. Note position and application of Electrodes.

It will be noted that the electrical axis of the heart theoretically extends downward and toward the left, following in a general fashion the actual position of the heart. Start-

ing just to the right of the sternoclavicular junction at an area corresponding to the junction of the superior vena cava and the right auricle, a line can be drawn downward and to the left of the apex of the heart. This line roughly divides the entire body into two portions; that part to the right, which includes the right arm and right leg, and the other, which includes the head, left arm, and left leg. Certain values have been rather accurately determined in regard to the difference of electrical potential as manifest by these portions of the body. For example, assuming X to be the unit of electrical energy developed by the heart and distributed to the various terminal points of the body, it will be found that the right arm can be conceived as minus 4X, the left arm as plus 2X, and the left leg as plus 3X.

It will thus be noted that theoretically, the addition of the unit strengths given for the right arm and left leg is the greatest of the three possible circuits. Clinically, it is found that the excursions developed by Lead II are the largest. When, however, the electrical axis of the heart is moved, either to the right or left, the values given for the left arm and left leg change, so that the circuit indicated by Lead II no longer gives the greatest units of strength, but that Lead I or Lead III become greater.

It has been noted that anatomical changes of the heart may change the electrical axis. In this connection, two well-defined conditions have been described. When physiologic or pathologic conditions arise in the cardiovascular system, sufficient to increase the size of the left ventricle, or the left side of the heart, the electrical axis is moved toward the left, giving greater unit values to Leads I and III than to Lead II. The theoretical fulfillment of this new relationship presents greater positive excursions in Lead I, and greater negative excursions in Lead III. In other words, the QRS complex representing ventricular contraction, will be found upright in Lead I, and downward in Lead III.

Such a change is known as *Left axial deviation of the heart.*

Where, however, the pathology which has taken place in the heart involves especially the right ventricle, the electrical axis of the heart may now move toward the right. Referring to our diagram again, we will note that the devia-

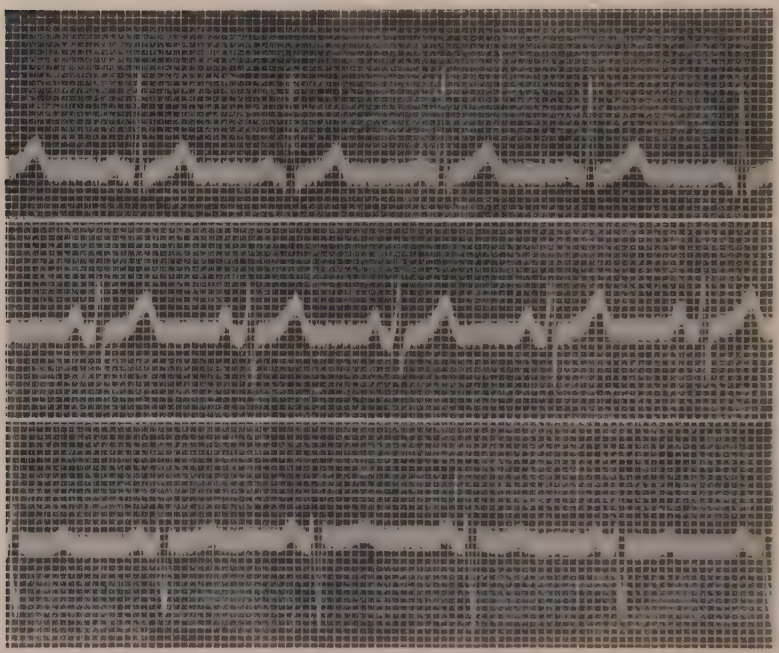


FIG. 5.—Left axial deviation. Note QRS complex in Lead I pointing upward and the QRS complex in Lead III pointing downward. (In this figure as in all of the following, unless otherwise indicated, the upper tracing is Lead I; the middle, Lead II; and the lower, Lead III.)

tion now gives greater negative values for Lead I, and greater positive values for Lead III. Clinically, this is shown by the downward excursion of the QRS complex in Lead I, and the upward excursion in Lead III. Such a change is known as *right axial deviation of the heart.*

The terms left axial deviation and right axial deviation have gradually supplanted the original nomenclature of

right and left ventricular preponderance. Other conditions than those dependent on ventricular change, have been found to present axial deviation. For example, enlargement

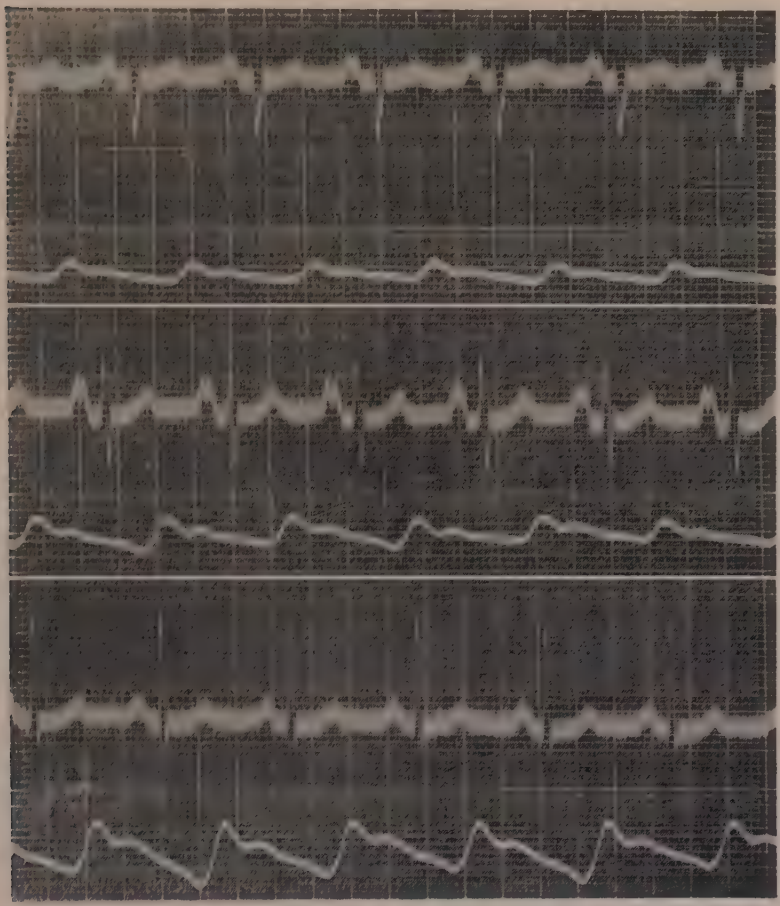


FIG. 6.—Right axial deviation. Note that the QRS complex in Lead I is pointed downward and in Lead III it is pointed upward. Simultaneous polygraphic tracings of the radial pulse have been taken with each of the three leads.

of the aorta, by changing the electrical axis of the heart, will give a left axial deviation, while the left ventricle may show little or no change at all.

From time to time, various terms have been used to describe these phenomena; left ventricular preponderance, left ventricular predominance, left axial rotation, and left axial deviation have been used interchangeably and a synonyms. It is better for the beginner to confine himself to the phraseology using axial changes of the heart, rather than to express these electrocardiographic findings in terms of anatomical changes of the heart. In other words, a scientific interpretation of such graphic records must not assume the character of a clinical diagnosis. This dictum follows the period of overenthusiasm engendered by the early investigators of the electrodynamic factors of the heart. Tempered by the only too frequent discrepancy between the postmortem table and the electrocardiographic findings, this overenthusiasm has rightfully given way to a strict scientific statement of facts.

CHAPTER II

THE ELECTROCARDIOGRAPH, ITS DESCRIPTION AND TECHNIQUE

THE ELECTROCARDIOGRAPH, ITS DESCRIPTION AND TECHNIQUE

The application of electrical methods to the study of the heart has formed one of the most important of recent advances in cardiology. This application was made possible largely through the work of Professor Einthoven of the University of Leyden.

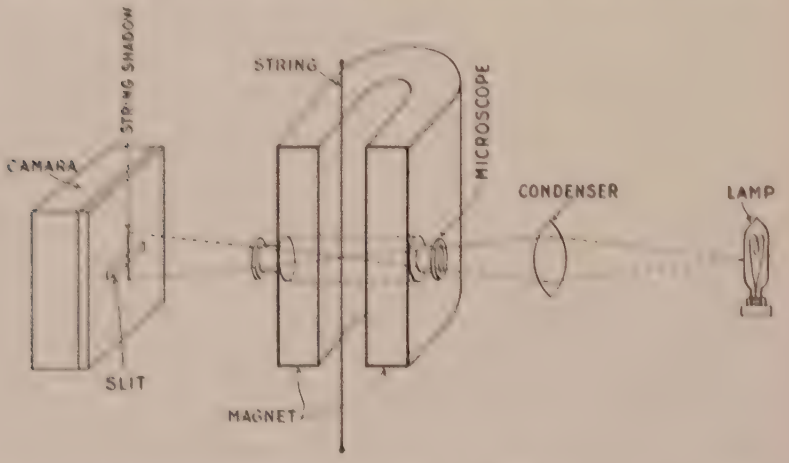
In the following pages an attempt has been made to present a complete, though concise, description of the string galvanometer and of the various auxiliary apparatus which have been evolved to enable the instrument to be used for electrocardiography.

Many advances have been made since the first instrument of Dr. Einthoven, and in designing the modern apparatus, the aim of most of the manufacturers has been to make it as simple and robust as possible, at the same time eliminating all complicated and unnecessary features.

It is well known that all muscle movements are accompanied by changes in electric potential of the active part relative to the passive part, the active part being negative, i.e., if a galvanometer is connected with a muscle under stimulation, by means of electrodes, a current will pass through the galvanometer from the electrode nearest to the passive part of the muscle to the electrode nearest to the active part of the muscle. If, therefore, a record of the electrical currents produced by the heart's action can be obtained, it follows that this must give reliable information regarding the various phases in the action of this organ.

Although the theoretical possibility of this had long been appreciated, it was not until Einthoven evolved the string galvanometer that it became a practical possibility. He introduced his new instrument, the string galvanometer, in 1903, and as its name implies, the vital part of the apparatus is a string or fibre. This string is made extremely fine, and,

when suitably mounted in a magnetic field, responds with a high degree of accuracy to the minute electrical currents of the heart. The actual movements of the string are very small and it is necessary to use a microscope in order to observe or record them. At the speeds and magnification used for cardiographic work the string must be strongly illuminated, either a specially constructed electric bulb or an arc lamp being employed. Fig. 7 shows diagrammatically



Diagrammatic representation of the Electrocardiograph

Fig. 7.

the arrangement for illuminating the string and for photographing the string movement.

The string is illuminated from the arc or electric lamp, the light being concentrated by the condensers. The beam is projected on to a cylindrical prism in the front of the camera, which focusses part of this into an intensely bright band of light in the plane of the sensitised film or paper, the light passing through a slit between the cylindrical prism and film. The string appears in front of the prism as a long vertical shadow about a millimeter wide, the part striking the cylindrical prism becoming a dark spot in the

band of light which falls on the film. Thus, if the film or paper is given a motion at right angles to the cylindrical prism, the whole length will be exposed except that portion which is hidden by the shadow of the string. The movements of the string, however, are in a direction parallel to the length of the cylindrical prism, and, as the instantaneous position of the string is indicated by an unexposed spot, a continuous record of these positions is formed on the moving film or paper. A series of such records are called electrocardiograms.

To produce the horizontal markings shown on the records, lines are engraved on the cylindrical prism at intervals across its width, these lines producing shadows which form lines along the length of the entire record. The vertical lines are produced by interrupting the focussed beam of light, so that for a short interval no light falls on the film as it is traveling past the slit, and in consequence a sharp line appears on the record.

As will be seen later, the action of the heart produces changes of potential at all parts of the body, and, as the extremities of the limbs are most readily accessible, it is usual to make electrical connections to the body through them. The connections to the limbs must be made by means of non-polarizable electrodes, otherwise considerable distortion of the curves is introduced.

Superimposed on the varying currents due to the heart there is usually a relatively large and fairly steady current, due to a potential difference caused by the glandular activities of the skin and known as the *Skin current*. It is necessary that this potential difference should be counterbalanced, and this can only be carried out accurately by introducing an equal and opposing potential difference. This operation is performed by means of the *Control board*, which also provides an arrangement whereby a definite potential difference can be applied to the string and the corresponding deflection measured and suitably adjusted.

Einthoven standardized to a sensitivity of 10 millimetres deflection for one millivolt, and this standardization is now almost universally adopted, thereby making electrocardiograms by different experimenters easily comparable. Provision is made in the control board for measuring the patient's body resistance and the galvanometer resistance, and a switch for making the various lead connections in the correct order is also provided.

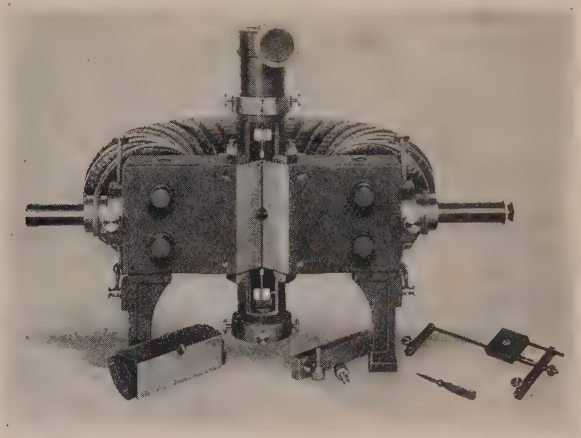


Fig. 8.—Hindle-Williams galvanometer, large model.

The *Galvanometer,* which is the essential part of all electrocardiographic outfits, is illustrated in Fig. 8, while Fig. 7 shows diagrammatically the principle of the instrument. It comprises a powerful electro-magnet between the poles of which an exceedingly light string is stretched. When a current passes through the string, it is deflected at right angles to the magnetic field. If the direction of the current in the string is reversed, the latter will move in the opposite direction. The deflections of the string are proportional to the strength of the magnetic field and to the current passing through the string, while they decrease with increasing tension of the string. By making the string as light as possible and using a very powerful electro-magnet, the sensitiveness

of the instrument can be made sufficiently high to enable the string to respond accurately to the extremely small and rapidly varying currents set up by the muscle movements of the heart. The string is usually made of silica, with a thin film of silver deposited on its surface. The length of the string is approximately 100 mm. and its diameter about .003 mm. The field coil is wound in two sections, and by arranging these in series or in parallel the galvanometer can be excited on either of two voltages, the standard winding being for 110 volts and 220 volts D.C. when a lighting supply is used, or 10 and 20 volts if accumulators are used. The energy consumed is about 60 watts in each case, with the coils in series or in parallel. Owing to the fact that the field of the galvanometer is nearly saturated, a considerable deviation from the standard voltage does not greatly affect the galvanometer sensitivity, but if the supply voltage is subject to sudden fluctuations it is advisable to employ accumulators to excite the galvanometer, as the fluctuations will introduce distorting effects in the cardiogram.

The movements of the string can be observed by means of a microscope, which passes through a hole bored through the centre of the magnet poles; by using a powerful source of light and concentrating it on the string by a condenser, the shadow of the string can either be thrown on a screen or photographed by means of a suitable camera, which latter is the method most usually employed in cardiographic work. A specially made optical tube of small diameter is used, so that the hole in the pole pieces through which it is inserted is correspondingly small, and the strength of the magnetic field and the galvanometer sensitivity are not seriously reduced in consequence. The string is completely enclosed in the string case (Fig. 9), which, being airtight, prevents the slightest disturbance of the string due to draughts, excludes dust and dirt and provides a mechanical protection which eliminates the possibility of the string being accidentally touched and broken. A milled head is

fitted to the string case and allows accurate adjustment of the tension and sensitivity of the string. The device for applying the tension is fitted with a safety stop which removes all risk of the string being broken through overtightening. The string case is held in position by a screw and spring. It is in consequence very easily withdrawn and replaced, with the certainty that after replacement the string will be returned to exactly the same position as before in the magnetic field. The position of the string is arranged as far as possible from the heat developed in the electromagnetic coils, thereby reducing to a minimum the temperature changes occur-

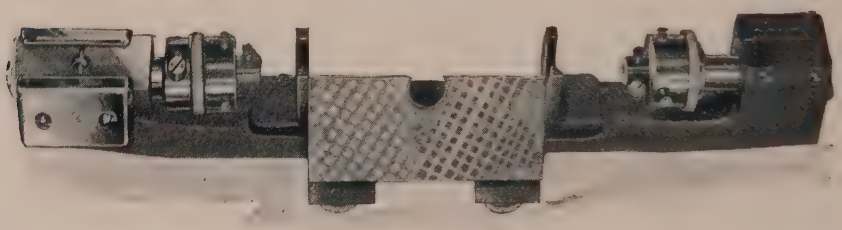


Fig. 9.—Hindle-Williams string case.

ing in the string case, and so giving the zero and sensitivity a high degree of constancy.

In ordinary use the string will last for a number of years, but it is convenient to have an easy means of inserting a fresh string in case of accidental breakdown. This is best secured by having a spare string case, with string already mounted, which can be substituted for the string case previously in use within a minute or two.

The Control Board provides in a simple form means for performing all the electrical operations required in taking electrocardiograms, viz:—(1) Making the various connections in correct order for Leads I, II, and III. (2) Compensating for the skin current, (3) Standardizing the sensitivity of the galvanometer, (4) Measuring the body resistance of the patient, (5) Measuring the galvanometer resistance. All

the contacts on the control board are enclosed to avoid any inaccuracies that might be caused by the accumulation of dust and dirt, and the switches are operated by turning the handles.

Fig. 10. Control board, Hindle-Williams electrocardiograph

Fig. 11.—Control board, Sanborn electrocardiograph.

The Camera may be used for both film or paper. Rolls of bromide paper about 200 feet in length are employed, the paper being made to travel past the lens by means of an

electrically-driven friction roller, and then fed into a box in which it can be removed for developing. Records of considerable length can therefore be obtained, and this fact makes the use of paper particularly serviceable for experimental work and for special cases requiring a record of long duration, e.g., cases of very occasional irregularity or paroxysmal tachycardia, where certain phases of the paroxysm are to be recorded.

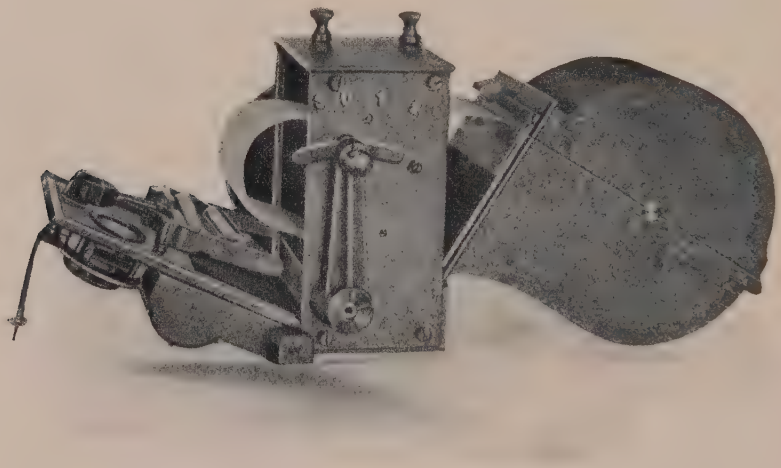


Fig. 12.—Hindle-Williams electrocardiographic camera.

The camera itself consists of three parts—a light-tight box in which the roll of unexposed bromide paper is contained, the camera proper containing the mechanism for carrying the paper past the slit, and a second light-tight box below the camera into which the paper is fed after exposure. The two boxes are removable, so that the upper one can be taken to the dark room for reloading, and the lower one can also be removed to the dark room for development of the exposed film or paper. The paper, after passing through a narrow slit in the bottom of the box carrying the unexposed roll, passes over a friction roller which is mounted on the

same shaft as the cone pulley. A loose roller presses the paper against the fixed roller, and as it passes over the face of the latter the paper is brought immediately behind the slit of the camera and so exposed. From these rollers it is fed by means of suitable guides through a narrow opening in the bottom of the camera into the removable box underneath. The opening through which the paper enters the box can be closed by a sliding shutter, and a knife severs

Fig. 13.—Hindle-Williams time marker.

the exposed length of the paper from the main length and enables it to be removed in the box to the dark room. The camera is connected to the motor by a driving belt and the speed regulated by increasing or decreasing its tension.

The *Time marker* used to give the time lines on the photographic records obtained with the electrocardiograph consists of a small electric motor controlled by a vibrating tuning fork and carrying a spoked disc. The tuning fork is suspended by a spring and hook below the cardiograph table, and maintained in vibration electrically by means of a small

electro-magnet excited from the main lighting circuit. When the current flows through the electro-magnet, the tuning fork is attracted towards the magnet until the motion of the tuning fork breaks a spring contact and cuts off the current

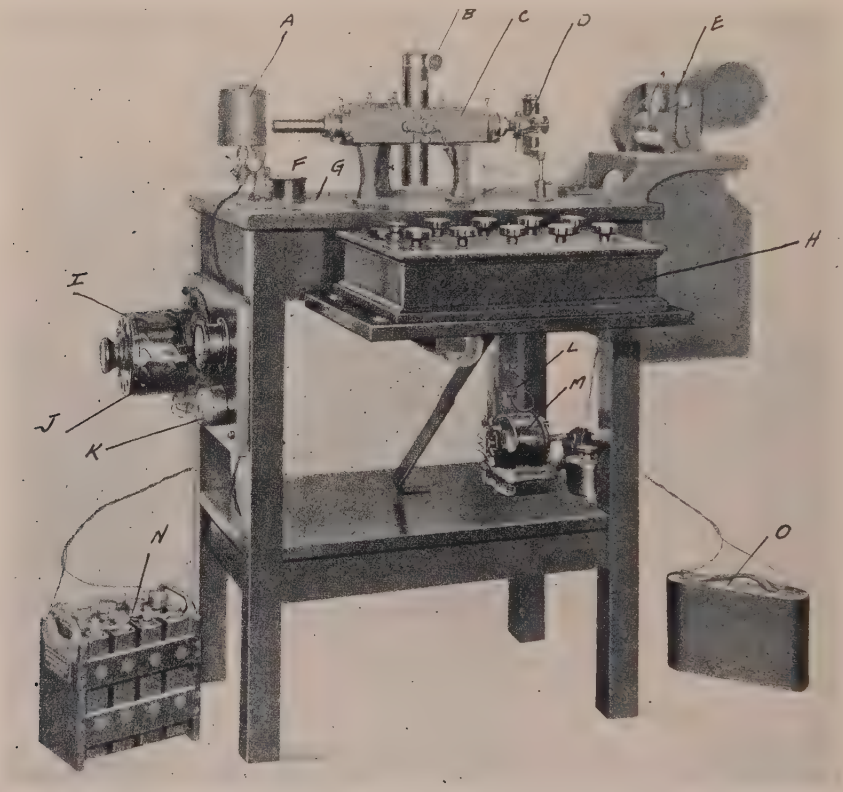


Fig. 14.—Hindle-Williams electrocardiograph. Office type. A. Lamp. B. String case. C. Galvanometer. D. Tine marker. E. Camera. H. Control board. L. Tuning fork. M. Motor. N. Storage battery. O. Dry cell battery.

from the electro-magnet. The tuning fork then moves back until the circuit through the electro-magnet is again completed. In this way the tuning fork is kept vibrating at the rate of 50 vibrations per second. The armature of the motor has ten teeth and one tooth is attracted towards the elec-

tro-magnet every time the current passes through the coils, which means that the armature will make five complete revolutions per second, and once it has been started at this rate it will continue running for hours. The spoked disc which rotates with the motor has a number of evenly spaced projections around its periphery, every fifth one being about twice the width of the others. The motor is so set that the projections on the disc intercept the light from the eyepiece of the galvanometer. The result of this is to cause lines at regular intervals on the record, due to the momentary non-exposure of the paper. The thicker spokes make wider lines than the others, so that the time lines are divided into groups of five, which is found a convenience in measuring up the records. Since the disc rotates five times per second, the thick lines mark fifths and the thin lines twenty-fifths of a second.

Since the development of the original instruments utilizing the principle of the Einthoven string galvanometer, various improvements have been added from time to time by those interested in their manufacture. In this connection must be mentioned the apparatus developed by the French engineer Boulitte. This latter machine eliminates the compensating current required for reducing the specific effect of glandular and skin electrical activity. This has been done by employing the theory of the Wheatstone bridge in dividing or splitting the electric current instead of neutralizing it by a series of choked resistances. Stripped of its complex technicalities this means in practice that no attention need be paid to the so-called "skin currents."

In addition to this, the Boulitte machine utilizes the principle of positive action in its photographic recording equipment; instead of having the photographic paper or film passed through the camera pulled by friction rollers it is carried by means of sprocket wheels engaging in marginal perforations of the film or paper. This principle has been adopted from the moving picture apparatus. It is said

that the accuracy of the completed records, so far as the time element is concerned, is considerably enhanced by this method. Another feature which commends itself to those

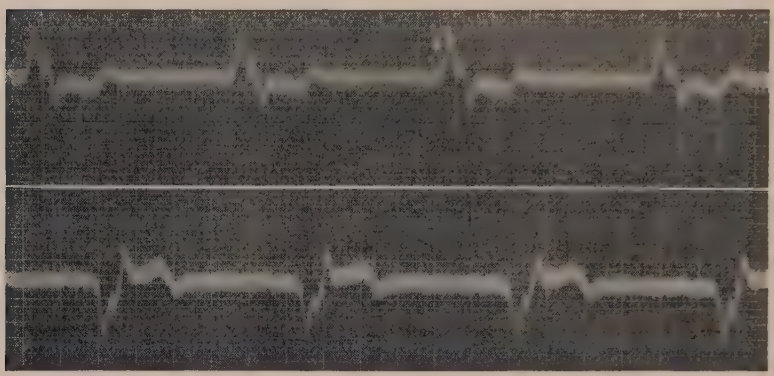


FIG. 15.—Double exposed film showing the first and third leads taken on the same film

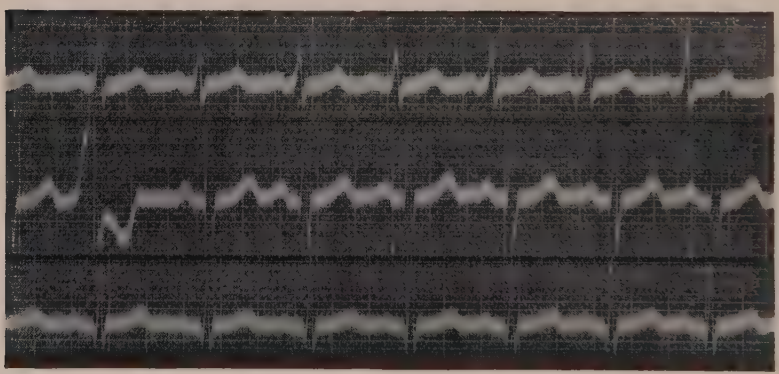


FIG. 16.—Triple exposed film showing the three leads taken on the same strip, but not photographed simultaneously.

interested in the experimental phase of electrocardiography in addition to its practical clinical aspect is the fact that the film or paper can be turned backward and double or triple exposures made. In other words, two or three leads

may be recorded on the same film; while this principle has been used on the photographic plate equipment of the older machines, the length of the tracing has been limited to the size of the plate employed. This plate is only six inches long so that the tracings which are obtained are frequently too short for clinical use. On the other hand, the Boulitte machine permits the taking of records from 18 to 20 inches long.

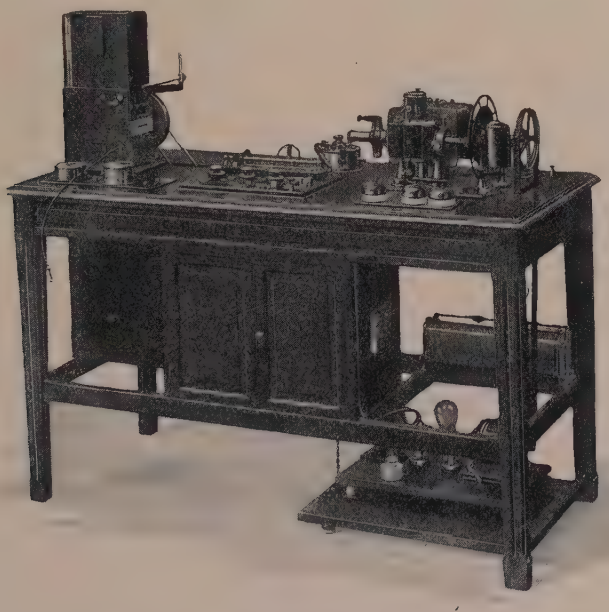


Fig. 17.—The Boulitte electrocardiograph.

Another departure in the French machine is the synchronisation of time-recording device with the rotation of the camera. This has been made possible by an electrically controlled governor which regulates the speed of a specially designed motor. The time marking device is therefore merely a rotating wheel with five spokes similar in effect to the Hindle-Williams and the Cambridge machines. Where a

110 volt direct current is available, no storage batteries are necessary in the manipulation of the Boulitte machine; a system of balanced resistances reduces the commercial line current to the various voltages used by the different parts of the machine.

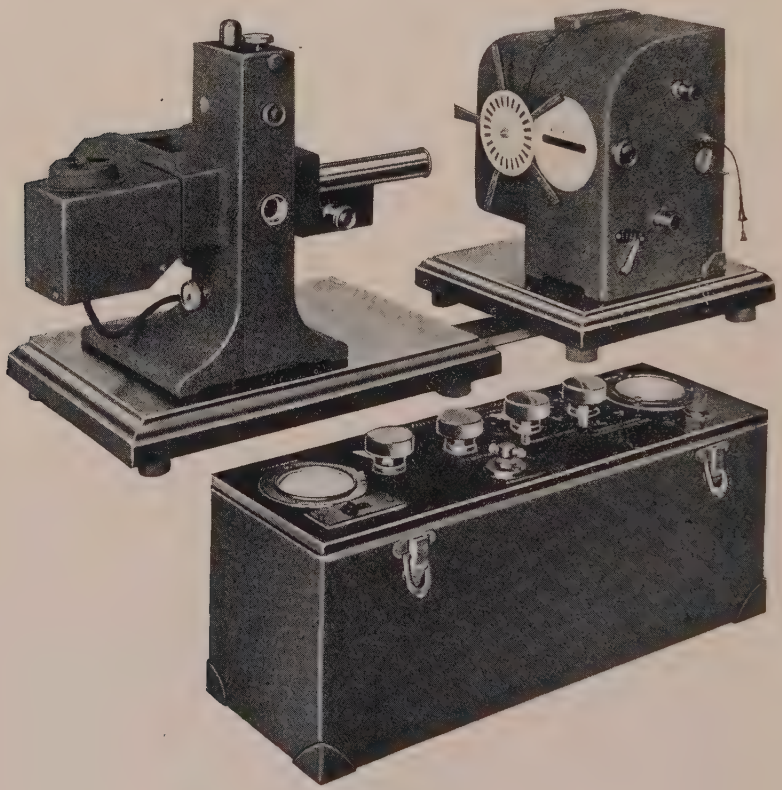


Fig. 18.—Sanborn electrocardiograph.

The Sanborn instrument is essentially the same both in principle and operation as the Hindle-Williams and Cambridge instruments. This instrument has reduced the very complicated electrical and optical systems to a minimum, so that the entire apparatus is small and easily transportable. In addition to this the time recording apparatus

and the photographic equipment are activated by a phonographic spring motor. This motor is delicately controlled by a ballistic governor and is very positive in action.

Of considerable interest are the newer models of the German electrocardiograph developed by the Siemens & Halske Company of Berlin. This machine has modified the original Einthoven galvanometer in two ways. First,

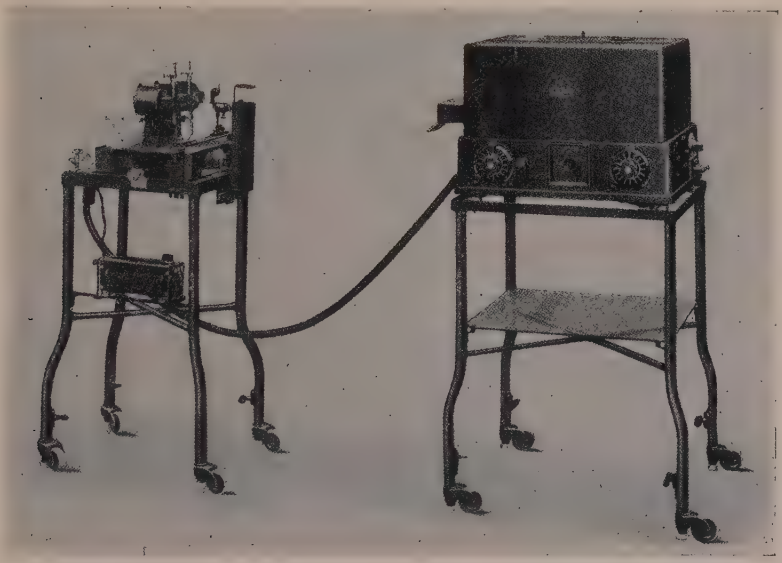


Fig. 19.—The Siemens and Halske electrocardiograph.

several instead of one magnetic coils are used; this has made possible the development of two or three magnetic fields instead of one. In effect, this means that two or three strings can be used at the same time, or in other words, two or three leads can be taken simultaneously. From a technical point of view, however, the simultaneous registration of the three leads presents many difficulties, so that in actual clinical practice only two leads are taken. The advantage of the simultaneous registration of two significant leads is obvious; extrasystolic phenomena and altera-

tions in the various waves can be studied with greater accuracy.

The second great change is that made in the modification of the Einthoven "string;" instead of this being the delicate and fragile fibre used in other machines, it consists of a fine wire with a small coil. Mounted on this coil is a minute mirror. In action, the electrical energy of the heart activates this small moving coil, turning it within the magnetic flux of the galvanometer field instead of moving

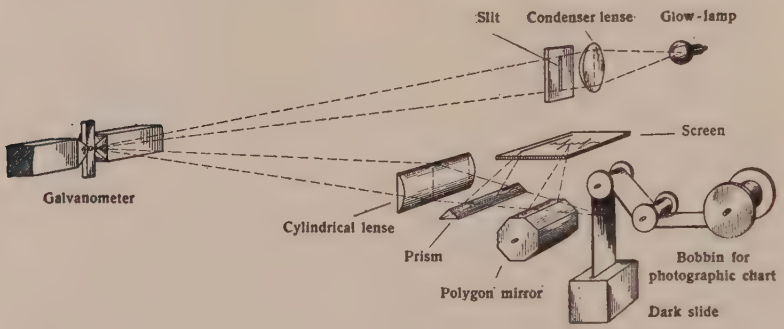


Fig. 20.—The Siemens and Halske electrocardiograph schema.

it at right angles as in the ordinary string instrument. A beam of light is focused on this delicate mirror and its slight rotation is recorded on the photographic equipment, greatly magnified. The advantage of employing this modified string is that it reduces the fragility hazards of the ordinary fibre.

The photographic equipment of the Siemens machine has several unique features; one of these is a revolving polygonal mirror which reflects the beam of light returning from the galvanometer on to a ground glass plate. In this way the electrocardiographic record can be easily visualised. The timing device of this machine is a vibrating steel reed, electrically activated.

An electrocardiograph differing radically both in principle

and operation from the string galvanometer type was recently introduced by the Victor X-Ray Corporation of Chicago. The apparatus does not use a quartz string and instead of the shadow of a moving string, the recording element is a beam of light reflected from a moving mirror. Heart voltages are amplified by means of a three-tube

Fig. 21.—The Victor electrocardiograph.

especially constructed amplifier to such strength that a sturdy, rugged galvanometer can be used. The apparatus is portable and can be carried about without danger to the internal parts. The accuracy or sensitivity of the equipment is constant regardless of outside conditions. Low or high body resistance does not affect the apparatus, and no compensation or adjustments are necessary, and no neutralization for skin voltages or currents need be taken into con-

sideration. Overshooting of the galvanometer is impossible under any conditions. The apparatus can be operated in broad daylight. Incorrect manipulation of the controls will not destroy any of the parts of the equipment. Since the apparatus accuracy or sensitivity is not variable, it can be standardized without the patient in the circuit, as well as with the patient in the circuit. There is no necessity for

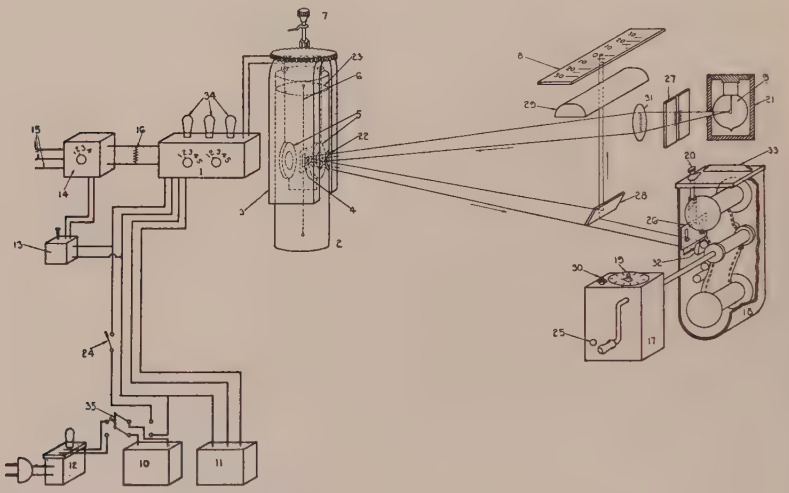


FIG. 22.—Schematic diagram of Victor electrocardiograph.

focussing the light beam since once this is set in the factory, it never changes. It is possible to make a record on any patient who can relax, regardless of the resistance, weather conditions, or variable skin voltages, with the same degree of accuracy. Because of the simplicity of operation, no trained technicians are necessary.

Since this machine is such a radical departure from the usual types of apparatus employed, and because of its recent introduction into clinical medicine, very little information is at hand in regard to its use and advantages or disadvantages. From time to time certain objections have been

raised; the first is that the apparatus utilizes no time marking device. The manufacturers state, however, that this is unnecessary, as the mechanism is so accurate that the time element may be added afterwards by either photographing a time grid with the original record, or the tracing can be viewed by being placed over a specially prepared ruled paper, containing the various time and voltage elements. Many physicians feel, however, that such accuracy is only assumed and should certain changes take place in the speed

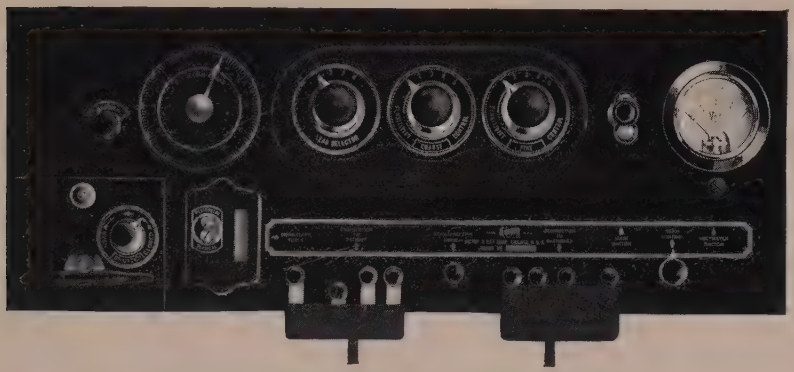


Fig. 23.—Control board, Victor electrocardiograph.

of the recording photographing equipment, it could not be discovered. Another objection has been raised in regard to the size of the photographic film which is used. This film is the ordinary moving picture film and in many cases it would prove to be too narrow when high voltages occur. Moreover, this narrow film does not permit the use of accessory curves like that of the radial or jugular where these may be necessary. On the other hand, the instrument is the most easily portable and the simplest to operate. In the principle of audion bulb modification of the heart currents may lie the future development of simplified cardiography.

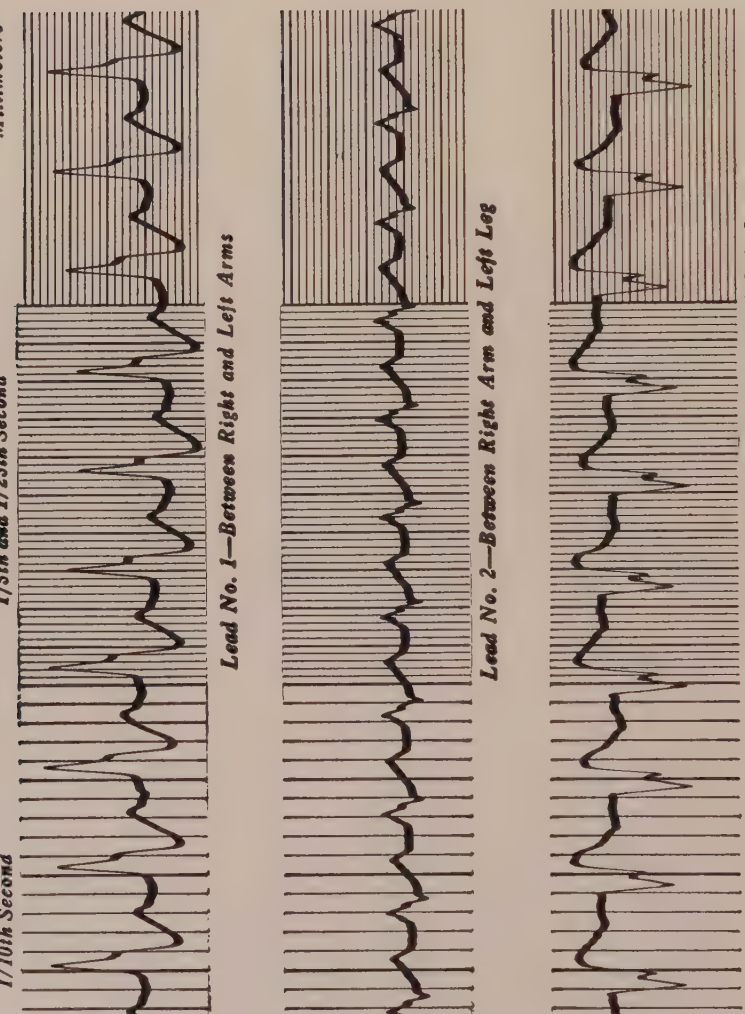


FIG. 24.—Time marking grid, Victor electrocardiograph.

LOCATION OF ELECTROCARDIOGRAPHIC EQUIPMENT

The vagaries of instruments used in micro methods are only too well known by those employing them; the electrocardiograph is no exception. For this reason the location of the electrocardiographic equipment in the building where it is to be used is of especial importance. Where the apparatus is used exclusively in a physician's office located in the ordinary home, the problem may be very simple and no particular precautions may be necessary. Where, however, the apparatus is to be employed in a modern hospital or office building, the problem of induced currents from adjacent steel structures, power lines, elevators, electric railways, X-ray plants, and radio stations, may assume proportions that often render the instrument unfit for use.

In general, many of these difficulties may be greatly reduced and sometimes eliminated entirely by efficient grounding of the apparatus; too much attention can not be paid to this detail.

Many times, however, induced currents are not so readily removed: upon meeting with such difficulties we have found that the theory of "screening," as adopted by the radio engineers, is perhaps the most effective method of eliminating such induced currents. This consists in surrounding the walls of the room as well as the ceiling and floor with a metal network, like chicken wire or other coarse metal mesh. This wiring can be effectively concealed under plaster, if such specifications are made while the hospital or building are under construction. Care must be taken to have all parts of the screening electrically connected and carefully grounded. Where the electrocardiograph is to be placed in a room already completed, the screening may be placed directly upon the walls, ceiling, and floor, and covered over by wall paper or other suitable material such as imitation

tile or burlap. The floor screening may be covered by linoleum. Such screening has been found to render the most delicate instrument entirely free from induced currents, regardless of how near the offending electric circuit may be.

Where free choice is permitted in locating the electrocardiograph in a room, it is preferably placed as far from a window as possible. First, because of the ever present hazard of the open windows where rain, moisture, and dust, may damage the exposed parts of the instrument; secondly, inasmuch as the apparatus employs a photographic device, darkness or semidarkness is desirable.

LOCATION OF THE PATIENT

In recent years the problem of transmitting the electrical energy developed by the heart from the individual to the electrocardiograph has been much simplified; it is no longer necessary to have the patient within the proverbial "arm reach" as advocated originally by Waller. In fact, there appears to be no reasonable limit to which the patient may be away from the instrument, actual tracings having been taken over an experimental cable at a distance of 25 miles. In hospitals and clinical practice it may in fact be desirable to have the patient located at some distance from the equipment so that movements within the room are limited as much as possible. In the hustle and bustle attending a busy cardiac clinic many accidents may occur as a result of unavoidable traffic in and through the electrocardiographic room. Moreover, in certain timorous individuals the sight of the apparatus with its equipment may induce nervous reactions that render the examination unprofitable.

For these reasons we believe that in so far as it is convenient, the patient should be placed in a room separate from the apparatus. In hospitals the problem becomes even more simplified, for a central electrocardiographic station can be constructed and cables led to the various floors and

rooms of the building, terminating in secondary stations or outlet boxes. These secondary stations can be placed at convenient points and connected with the patient by flexible cords. Considerable experimental work has demonstrated that certain precautions must be observed in planning such an electrocardiographic circuit. The cables carrying the heart current must be protected in the same fashion as the electrocardiograph itself from all possible sources of induced current. As a matter of fact, these extension cables may suffer more from the adjacent parallel power lines than the equipment. The proximity of telephone wires, signal systems, and bell circuits must be carefully considered. In addition to these possible sources of disturbance, the total resistance of the extension circuit must not be excessive. Increasing the resistance from the patient to the apparatus may entirely dampen the patient's record. This was found to be the case in an exceptionally well equipped institution when tracings were attempted upon patients on the seventh floor and above. The actual resistance of the extension service should therefore be carefully considered by those contemplating the building of new hospitals equipped with such service.

Such extension circuits may contain in addition to the three cables carrying the three leads also another pair of wires for telephone or signal service, so that the attendant at the bedside may indicate to the technician that the patient is properly prepared, and also that the operator may let the bedside attendant know that he has finished taking the record. Such additional signal service, while facilitating the taking of tracings from distant points, is not without additional hazards. It is not advisable to have the telephone circuit within the same common cable with the leads because induced currents are readily produced. Where such telephone service is installed, the wires carrying the circuit should be outside of the three wire cables carrying the leads, both of these cables, viz., the three wire circuit and the two

wire telephone circuit, should be enclosed in metal casings which are grounded from time to time.

In general office work, however, no such elaborate systems are necessary. If the patient is placed in the room adjacent to the apparatus, a metal armored cable containing the three leads may be drawn through the wall without further protection.

POSITION OF THE PATIENT

From time to time various investigators have suggested that one position or another is most suitable for the taking of tracings. Special chairs, tables, and couches of various types have been described and recommended. In general,

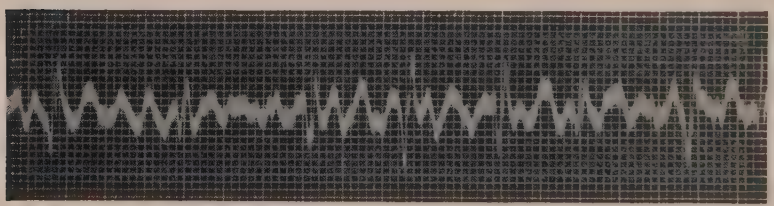


FIG. 25.—Record showing involuntary muscle oscillations due to patient assuming an uncomfortable position.

we have found that no emphasis can be placed upon the position of the patient, in taking tracings. Certain rules may, however, be formulated; first and foremost the patient must be comfortable, for absolute quiet and perfect rest are absolutely essential, if good records are to be obtained. If the patient is forced to assume a strained and unnatural position, muscle reactions may tend to obscure the tracing. One such illustration may be illuminating; a woman suffering from advanced auricular fibrillation, and with some degree of decompensation and dyspnea, was forced to lie almost horizontally upon a certain type of electrocardiographic table; her unconscious straining and

the resultant involuntary muscle action caused the record to show oscillations suggesting in every respect a so-called coarse circus movement. When the patient was permitted to sit upright in a chair, and when she became more at ease, a second tracing was taken showing no circus movement at all.

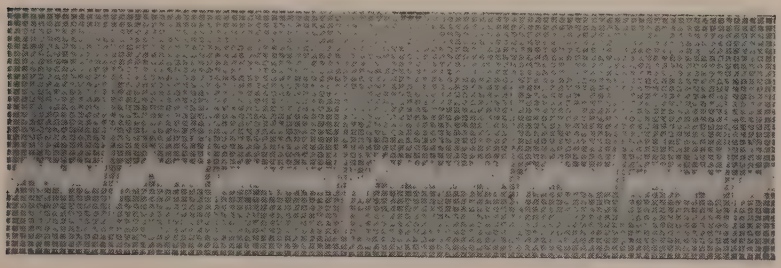


FIG. 26.—Same patient in a comfortable position.

In our experience we have found that most cardiac patients prefer to assume a sitting posture rather than a reclining or semi-reclining position. For this purpose any sturdily built comfortable chair can be used. The old fashioned Morris chair unquestionably is the best suited for this purpose, but the so-called billiard chair or ordinary armchair can also be employed. If the clinic is concerned only with ambulatory patients and, where dyspnea upon lying down is absent, a low couch is also suitable.

We can not censure too severely the high operating room type of table which is found in many physicians' offices and clinics. To expect obese and dyspneic men and women to clamber up upon such a high table is cruel and unscientific, yet one frequently sees such examining tables in cardiac clinics.

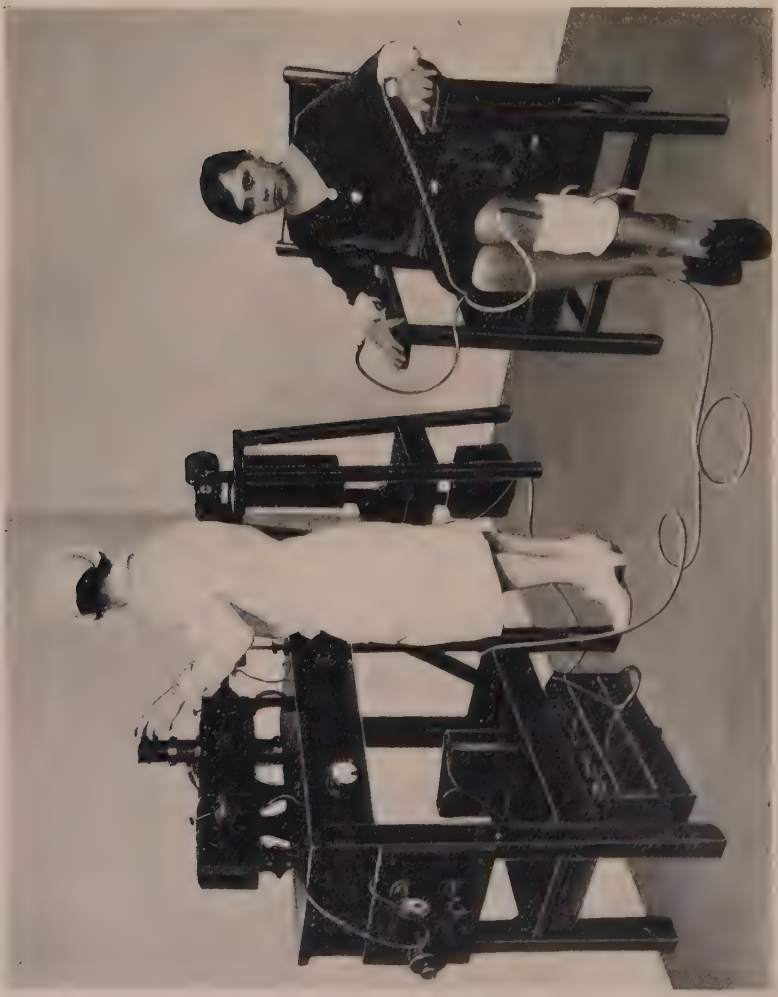


FIG. 27.—Photograph of chair with patient in position.

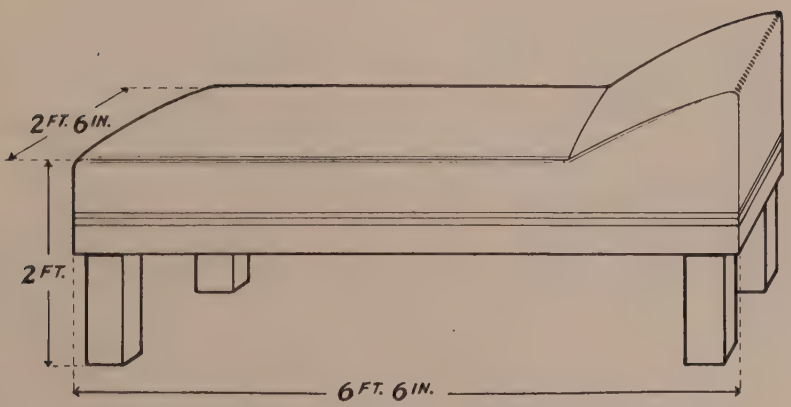


FIG. 28.—Low couch for severe cardiac patients.

PREPARATION OF THE PATIENT

Customs become universally standardized; out of the multitude of suggestions made in the last decade in regard to the application of the electrodes, probably only two have survived in most clinics. The problem of securing the electrical energy of the heart's action at its maximum involves a consideration of many physical and biological factors; these factors, for simplicity's sake, can be reduced to the facts of electrical phenomena. As has been indicated before, the selected points of application as terminals of the cardiac current have been determined, because the cross section diameter of the point renders it the most electrically active of any place on the body. The theory of electrical transmission states that energy is transmitted upon the surface of a conductor; where the conductor is extensive, the volume of electrical energy upon any one area must be less than when the conductor has less surface. This law applied to the human body would indicate that the wrists and ankles are the most suitable points of contact. The problem of the electrocardiographer is to obtain and to lead off from these three selected terminals the electrical energy of the

heart with as little loss as possible and with a minimum of distortion. For this reason it is necessary to make as complete an electrical contact as is possible; to this end, all grease and oil which are non-conductors must be removed from the skin, and some electrolytic solution well borne by the skin must be applied.

A whole gamut of electrolytic solutions have been tried, but the most common now in use are zinc sulphate and sodium chloride in saturated solution. The electrode itself has undergone many changes; from the simple metallic fillet of Vaquez to the very complicated non-polarizable electrodes of Price. In general, any metallic band or cuff can be used. A cloth or wide bandage saturated with the warmed electrolyte is placed either under or over the metallic band and wrapped snugly around the wrists and ankle. Every technician develops certain fine points of technique; a few of these may be enumerated with profit to the beginner: the solutions are better withstood by the patient if they are warmed. Cold electrodes may cause involuntary muscular tremors in susceptible and debilitated individuals; if the skin is gently but firmly rubbed with the bandages before application, a better contact is made. This may be due in part to the cleaning of the skin of oily and greasy material and also to the vaso-dilatation that accompanies the friction. The electrodes must be kept scrupulously clean; points of corrosion may cause polarizable currents. All contacts and clipping points must be bright and firmly attached, sand paper or emery cloth should be a part of the technician's equipment. Neglect of these items, small though they may be, may result in poor tracings.

The second method of securing the leads is by immersion of the hands and left foot into electrolytic solutions. This method is extremely simple in application; certain non-metallic basins or jars are filled with warm solution and electric contact is made from the jars to the instrument. The

patient merely places his wrists and foot into the containers and the tracing obtained in this fashion. This method enjoys great vogue in England and upon the Continent. In this country, however, it has not met with such popular favor, as it requires the removal of a shoe and stocking, the wetting of the foot, and the subsequent inconvenience of drying it. In addition to this, certain old and debilitated patients fear "catching cold" after this procedure, and in our experience we have found it not so well adapted for clinic use as the cuff electrodes. Moreover, the immersion method is not suitable for bed patients.

So far as the actual taking of records is concerned, we shall have very little to say, as each type of apparatus has its own peculiar method of operation and can be readily learned after some practice in any electrocardiographic laboratory. A few remarks may be made, however, in regard to problems met with in all types of apparatus.

Inasmuch as the electrocardiograph is a device for recording the shadow of a moving string upon a moving photograph film, the best results can only be obtained when maximum light and maximum shadows are produced; vague and indistinct registration of the string's shadow may render a valuable record worthless. All electrocardiographic equipment is provided with a system of lenses whose sole purpose is to concentrate light and to render the shadow of the string clear and sharp; string cloudiness and string halos are usually the result of improper light control, although, as has been recently pointed out by us, such indistinct and blurred records may be due to microscopical dust adhering to the fibre. In the latter case no amount of lense focusing will ever be able to make the shadow of the string sharp and distinct. It should not be necessary to point out that cleanliness of the lenses is absolutely essential; small particles of dust or condensed moisture may obscure the passage of light through the lenses and thereby render the record indistinct.

Aside from these details, the electrocardiographer must make himself thoroughly familiar with every part of his machine, so that he may instinctively turn to the point of trouble as soon as it occurs. Like automobiles, each individual apparatus develops its own peculiarities and the good technician is the one who learns how to take care of them. It might be pointed out here that the electrocardiograph is unquestionably *a one-man machine* with all that the suggestion implies; better and longer service is invariably obtained from those instruments where there has been but one operator.

IDENTIFICATION OF RECORDS

Where more than one record is being taken at one time, the problem of identifying the individual records as well as the respective leads may assume great importance. The danger of interchanging records and leads must be amply safeguarded and more than one method of identification should be employed. All electrocardiographic equipment has some device for photographing upon the record a number or a name. These should always be used in addition to a simple mark devised by the technician himself. The simplest of these may be just the passage of the hand across the path of light, thereby leaving an unexposed line upon the film; for the second record of the series two bars may be made, and for the third three bars. A standard policy of taking the three leads in consecutive order should never be abandoned. While it is true that the experienced cardiologist can readily identify most unmarked leads, not infrequently considerable confusion may result when the leads are not indicated.

LENGTH OF RECORDS

The expense of running an electrocardiograph is usually focused upon those parts which need constant replacement; into this group comes the film or photographic paper. Most technicians tend to be as economical as possible with this material and justly so, because of its extensive use. False economy in this regard, however, may prove costly because of the worthlessness of the records obtained. In routine clinics or office practice, where large numbers of patients are examined, a liberal record should be made upon the first examination; irregularities noted clinically should be recorded if possible. Much film, however, may be lost in attempting to photograph an unusual phenomenon occurring less than once per minute; every technician's waste basket is filled with such useless tracings. In order to eliminate this needless waste as much as possible, the following procedure may be of value. If the excursions of the string are carefully studied the irregularity can be noted without photographing it; with a watch in hand, its recurrence can be approximately timed as many irregularities recur with a definite frequency. This is especially true of extrasystoles. If it is found for example, that the irregularity occurs once in every three or four minutes it is worth while to study the phenomenon accurately, in this way the camera may be set in motion just before the irregularity is anticipated and it may be thus recorded on a minimum amount of film. It is advisable to take the first tracing of a case especially long so that enough of the material can be secured for adequate study; subsequent tracings may, however, be very much reduced. We have found that not more than one foot of film need be used for the entire three leads, each lead being about four inches long. Certain exceptions must be made in instances where the pulse is unusually slow or where irregularities must be recorded again.

Another method of conserving film and space is that ac-

complished by triple exposing the same film, so that each of the three leads are photographed on the same film. This requires a special device permitting the exposed film to be rolled back into the camera. All cameras which use regulation moving picture film can be employed for this purpose.

FILM OR PAPER?

Every beginner in electrocardiography is somewhat confused as to whether photographic film or paper should be used. In our experience, the answer must always lie in the use to which the tracings are to be put. Outside of the difference in expense, for film costs approximately four times as much as paper, the question of additional labor is important—the use of film requires the handling of the material twice. We believe that where the record serves no other purpose than to make a diagnosis, or where it is a normal one, paper should be used. Where, however, the records are of unusual interest and present some rare finding, or where the phenomenon is of a transient nature, photographic film is the medium of choice so that any number of reproductions can be made for distribution or study. The use of photographic film involves two distinct processes; first, in the handling of the film itself, and secondly, in the printing and reproduction upon photographic paper. It has been found expedient in most large clinics to use photographic paper almost exclusively, even in rare and unusual cases, because the former danger of not being able to reproduce the record has been almost entirely done away with in the development of the new photostatic process which can reproduce the original tracing with almost at little expense as the developing of film. Only occasionally is it necessary to photostat such records. From time to time one hears the criticism made that the tracings are more easily studied when they are produced by the photographic method, where the string excursions are black upon a white

background in contrast to the photographic paper records, where the string movements are white upon a black background. This, however, must always be a question of taste, for there is no real difference in the discernibility of both types of records.

FILM DEVELOPING

We have used the words film and photographic paper interchangeably throughout this chapter, for the use of both materials is essentially the same except for minor

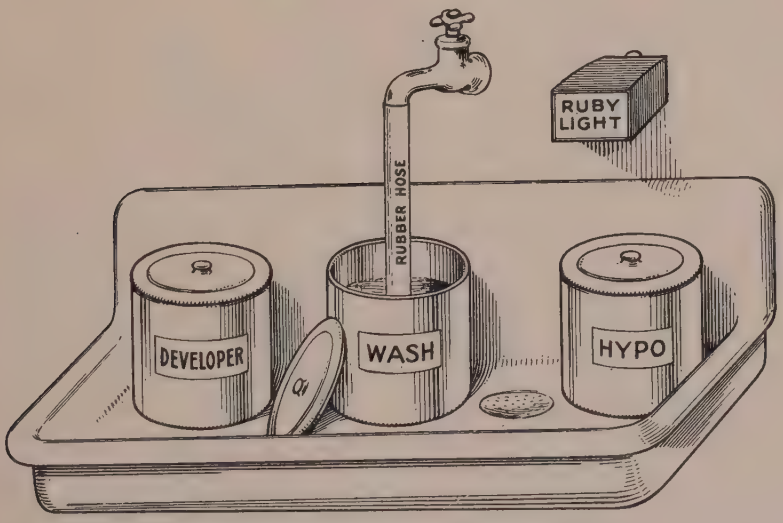


Fig. 29.—Developing jar arrangement.

details. The developing of electrocardiographic film differs in no way from the method used in amateur photography. The first essential is a photographic dark room where all light with the exception of the usual ruby lamp is excluded. The developing, washing, and fixing tanks need no special comment; these may be very elaborately constructed in a manner similar to X-ray equipment and the best of this

type are made of soapstone. On the other hand, the simplest tanks are just as satisfactory; for this purpose nothing is better than the old-fashioned five gallon earthen ware pickle jars with well fitting covers. These are cheap, impermeable, and easy to handle. Metal and even enameled vessels must be used with caution, as the chemical reagents used in the process readily corrode such tanks.

No further discussion in regard to the developing process need be added here save that good developing and fixing solutions must be obtained. It is again false economy to use cheap materials for this purpose, for the finished product can be no better than the developing chemicals can make it. Many painstaking records of unquestioned value are ruined or marred because of improper developing methods. Each type of film or paper has its own peculiar characteristics and the technician will do well in obtaining the developer most suited for his material.

Of great importance is the final washing of the records, more especially if they have been made upon paper, because even small quantities of the fixing solution may mar the surface of the paper after the records have been filed away. Photographic film is dried in the usual manner, but the paper should be rolled smoothly upon a ferrotype plate and allowed to dry slowly.

FILING AND STORING OF TRACINGS

The subsequent disposition of the tracings will of course vary with each individual case. So far as the paper records are concerned the problem is very simple; the records are cut to standard size upon a photographic trimming board and then glued or fastened to special charts or boards.

These sheets can then be filed with patient's history in a suitable cabinet. Where photographic film has been used, an additional procedure is necessary; when the film is

THE WITKIN FOUNDATION
FOR THE STUDY AND PREVENTION OF HEART DISEASE
BETH DAVID HOSPITAL

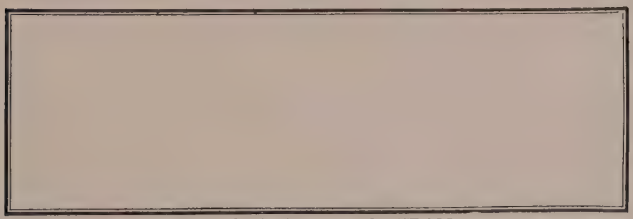
ELECTROCARDIOGRAPHIC RECORD SHEET

NAME ..... DATE .....
SERVICE ..... WARD .....

HEART STATION NO.

LEAD I. RIGHT ARM AND LEFT ARM

LEAD II. RIGHT ARM AND LEFT LEG



LEAD III. LEFT ARM AND LEFT LEG

Electrocardiographic analysis:

Electrocardiographic diagnosis:

(Signed) ..... M.D.
Cardiographer

FIG. 30 A.

48A Form No. 277

NEWARK BETH ISRAEL HOSPITAL

201 Lyons Avenue
Newark, New Jersey

Cardiographic Department

REPORT OF ELECTROCARDIOGRAM

No. _____
Ward _____
Clinic _____

Name _____ Date _____ Time _____

Resistance of Patient _____ *ohms* Resistance of String _____ *ohms* Dr. _____

Auricular Rate _____ *per min.* P-R interval _____ *sec.* Deviation of Electrical Axis _____

Ventricular Rate _____ *per min.* Q. R. S. _____ *sec.*

Deviation from Normal — Lead I _____
Lead II _____
Lead III _____

Electrocardiographic Interpretation _____

Taken by _____ Interpreted by _____

L. 1

L. 2

L. 3

FIG. 30 B.

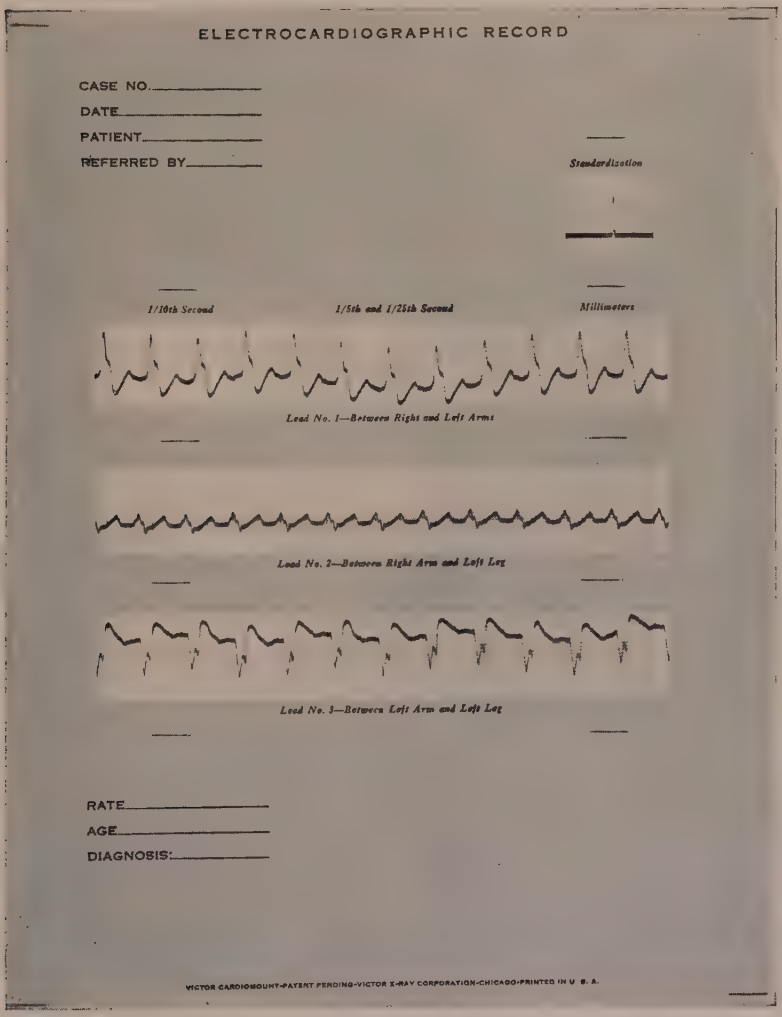

ELECTROCARDIOGRAPHIC RECORD
CASE NO.
DATE
PATIENT
REFERRED BY
Standardization
1/10th Second
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg
RATE
AGE
DIAGNOSIS:
VICTOR CARDIOMOUNT-PATENT PENDING-VICTOR X-RAY CORPORATION-CHICAGO-PRINTED IN U. S. A.

FIG. 30 C.

thoroughly dried, suitable high contrast photographic paper must be used in the printing process and then the prints may be handled in the manner described above.

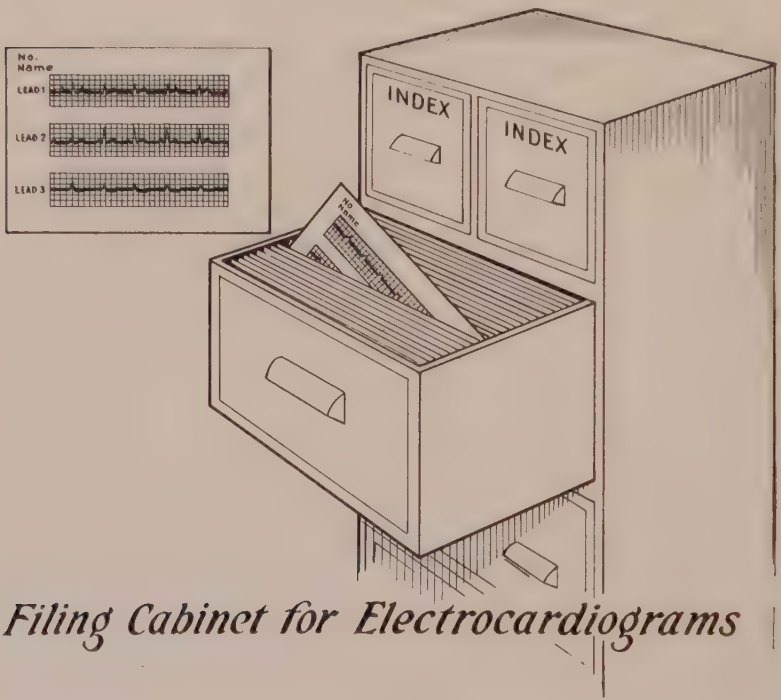


Fig. 31.

Because of the tendency to roll and curl, the films are best stored on spindles which are set upright from the bottom of a wide shallow drawer. Inasmuch as the film is inflammable and unless made from cellulose acetate, also explodable, these must be stored in fireproof filing cabinets.

CHAPTER III

DISEASES OF THE PACEMAKER

DISEASES OF THE PACEMAKER

Too little emphasis has been placed upon the rôle of the sinus node in the well-being of the individual, and insufficient attention has been directed toward this small group of cells which constitute actually the most vital point of the entire organism. There is probably no other area of equal size in the entire functional mechanism, that holds such an important place for the future of the individual. Conceive, for example, failure on the part of the pacemaker to initiate a stimulus for the cardiac cycle, and you will conceive instant death for the body. No other part of the animal structure is so completely safeguarded with devices to insure proper function. At the same time, the group of cells representing the birthplace of the cardiac cycle is subject to all of the influences, good and bad, that any other part of the body is subject. Indeed, diseases of the pacemaker itself, may not be an uncommon condition, although very little has been written about it.

As it has been previously stated, the function of the sinus node is to develop and release an electric impulse in the initiation of the cardiac cycle. The development of the impulse is dependent upon the special function of metabolism in which the nuclei of the pacemaker produce a change in their hydrogen-ion concentration at a given rate of speed. This process proceeds with the accuracy of a mathematical formula in which certain constants are maintained. When the change of hydrogen-ion concentration has reached a point of about Ph^{-5}, a difference of electrical potential arises between this new area of acidity and the surrounding cells. This difference of potential spreads like a wave over the auricular muscle and finally it reaches the auriculoventricular node.

The discharge of this electrical impulse occurs normally at the rate of 72 times a minute, but inasmuch as it appar-

ently comes within the laws of chemical reactions, it is affected by many conditions.

The simplest of these factors is that of temperature; heat increasing the reaction and cold retarding it. While the tachycardia or increase in heart rate in fevers follows the long established ratio of ten beats to every rise of one degree temperature, the actual process is not quite so simple, for a cooling of the pacemaker itself, is not accompanied by any such slowing of impulse formation.

For simplicity's sake, however, functioning of the pacemaker may be regarded as a purely chemical and electrical phenomenon guided by a certain complex metabolic regulation that involves neurogenic, endocrine, and vascular considerations.

Disturbances of the pacemaker can be divided readily into two groups; the first, those in which the impulse is generated too slowly, secondly, those in which the impulse is prematurely developed. Both conditions are seen in about equal numbers, and while premature generation of the impulses may be relatively innocuous, delay in the process may be accompanied by dangerous and often fatal results.

TACHYCARDIA

As described before, the pacemaker mechanism is susceptible and readily influenced by any condition which tends to alter the very delicately balanced metabolic process occurring within the cells of the sinus nodal tissue. Because of the close association with neurogenic structures, nerve impulses may have an important regulatory function so far as the impulse production is concerned. Vagal inhibitory action has been minutely scrutinized by many investigators, and its action in slowing the heart has long been recognized. Other neurogenic impulses arriving at the sinus node by certain ill-defined extracardiac pathways have been reported from time to time, and stimulation and depression

of these nerves have been found to alter the rate of the pacemaker.

Certain endocrine substances of which the thryoid is the most well understood, exert a specific effect upon the metabolism of the pacemaker cells and thereby increase their rate. Bainbridge demonstrated that increasing the velocity of the blood, entering the sinus venosus area, increases the rate of the heart beat. Segal showed that this increase in the pacemaker stimulus production was due to an increased blood supply to these tissues.

Physiological speeding up of the heart is probably the result of several factors. The normal heart responds to effort by increasing its rate of contraction. The need of more oxygen to the working muscles, the removal of waste products and CO_2, the liberation of adrenalin for the utilization of glyocogen, the compensatory influence of the thyroid, and an increased venous return to the right auricle, all contribute toward the acceleration of the heart beat. The actual effect upon the pacemaker cells by all these factors has been the subject of considerable study by many physiologists. In the normal healthy heart, these various factors are nicely adjusted, but small changes in any one of them may lead to well recognized pacemaker alterations. In the diseased heart especially, these factors may be readily ascertained.

While the limits of normal response to these influences may be variable, the well organized heart rarely beats faster than 140 beats per minute. Certain individuals, even after extreme effort, do not have a faster pulse rate than 120 beats per minute, while certain women may have a rate as high as 148 per minute. Cardiac rates faster than this, are usually due to some pacemaker disturbance. These rates, however, are usually not sustained, but are rapidly reduced as the physiological factors influencing the pacemaker are abated. Sustained pulse rates above 140 per minute, are usually due to a pacemaker disturbance. At

this point, it is interesting to discuss how fast the heart may actually contract. Clinicians are frequently confused in regard to this matter, and often report pulse rates of 200 and over. From a theoretical point of view, it can be determined that the normal cardiac cycle requires the following

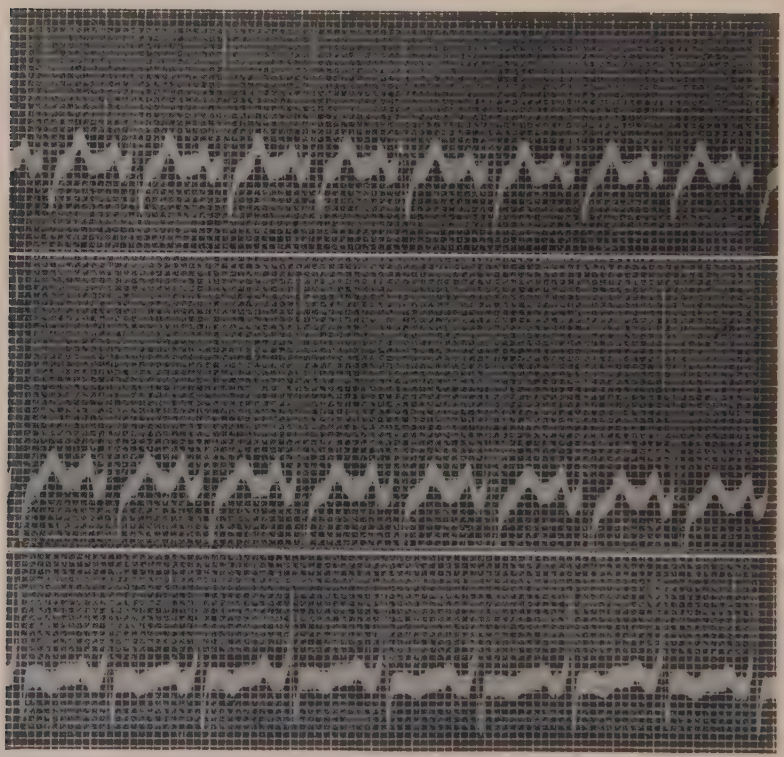


Fig. 32.—Normal sinus tachycardia after extreme effort. Rate about 140.

time intervals; the presystolic period represented by the P-R interval, measuring 0.18 seconds, the QRS complex measuring 0.08 seconds, and the R-T interval measuring 0.28 seconds, giving a total of 0.54 seconds for the duration of a normal cycle. There can thus be no more than 120 beats per minute without disturbing the integrity of the cardiac cycle. In cardiac rates faster than this, the T-P

interval, representing the diastolic phase, completely disappears, so that the T and P waves coincide. Under these conditions, the cardiac cycle can be shortened to about 0.30 seconds or 180 beats per minute.

Disappearance of the diastolic period from the cardiac cycle is not seen in physiologic tachycardias, and its occurrence should lead one to suspect a pathological disturbance of the pacemaker.

PATHOLOGIC TACHYCARDIA

Direct stimulation of the sinus nodal tissue may produce such a speeding up of impulse production, that the dias-

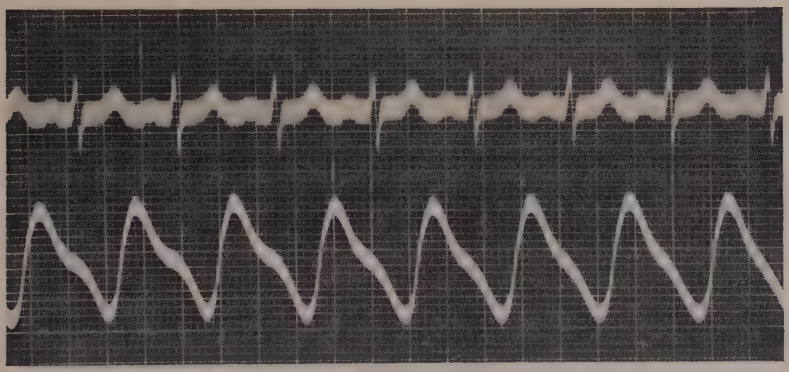


FIG. 33.—Thyrotoxic tachycardia. Woman, age 34, with marked clinical evidence of hyperthyroidism. Rate 124.

tolic period is lost. Such a condition may arise in hyperthyroidism; here the process is apparently explainable, as it has been very definitely established that the metabolic process is enhanced by thyroidal substance. Toxic substances produced as the result of bacterial infection, may also give rise to such speeding up of impulse production, although never to the same extent as the thyroid. The increase in the pulse rate in such infections may be due in great part to the pyrexia that accompanies this disease, but certain types

of pneumonia, influenza, tuberculosis, and malarial fever, may give pulse rates faster than the theoretical ratio of 10 to 1.

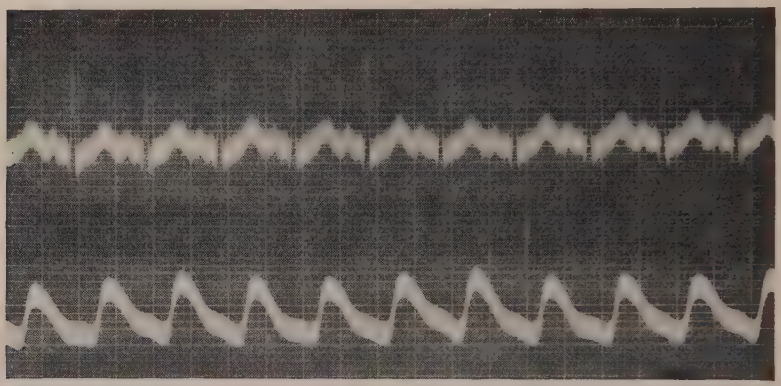


FIG. 34.—Sinus tachycardia occurring in a man 26 years old, during an attack of acute bronchopneumonia. Temp. 104.2°. Resp. 44. Rate 148.

Electrocardiographic studies of such pathologic tachycardias show a complete coalescence of the P and T waves.

NORMAL BRADYCARDIA

While the speeding up of the pulse rate is limited to mathematical considerations, the slowing of the pulse is made more complicated by certain life-saving mechanisms within the heart itself. The lower limits of the normal heart rate is open to some question. Sex, age, environment, general physical condition, and climatic conditions contribute to any definition of the lower limit of the normal pulse. In general, it may be said that pulse rates as low as 56 may be considered as normal.

PATHOLOGIC BRADYCARDIA

Inhibition of the metabolic process occurring in the pacemaker cells, may be the result of increased vagal effect.

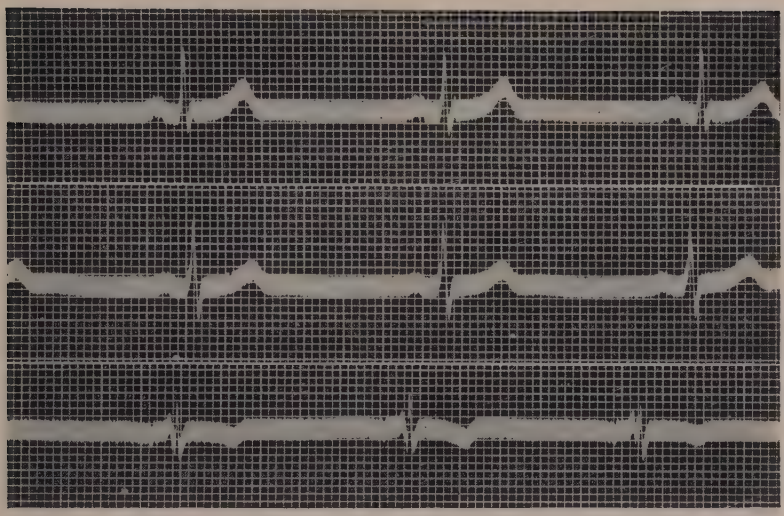


FIG. 35.—Normal bradycardia. Athlete, age 22. Rate 46.

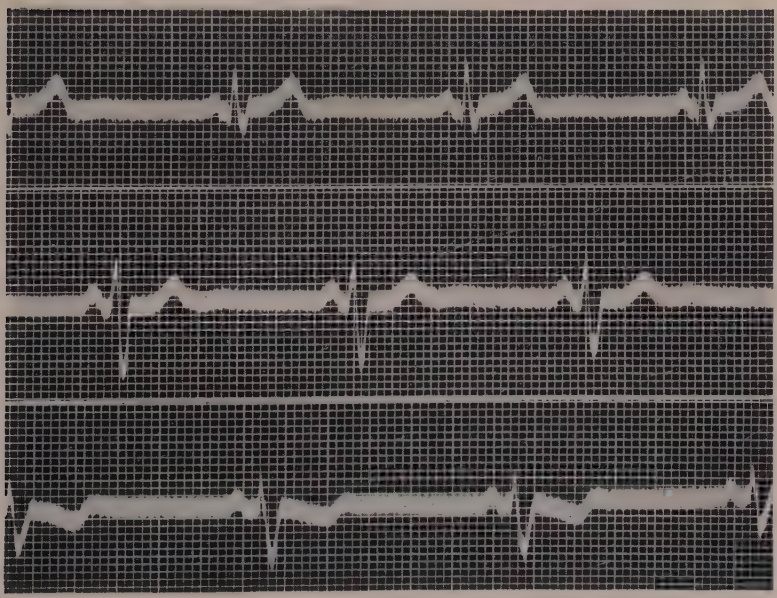


FIG. 36.—Bradycardia in a man of 48. Symptoms of brain tumor. Rate about 58.

Such conditions may arise in cerebral pathology, such as brain tumors, hydrocephalus, and concussion. Certain toxic substances, like those found in typhoid fever and influenza, may reduce pacemaker activity. Cholemia is very frequently a cause of bradycardia lower than 56 per minute.

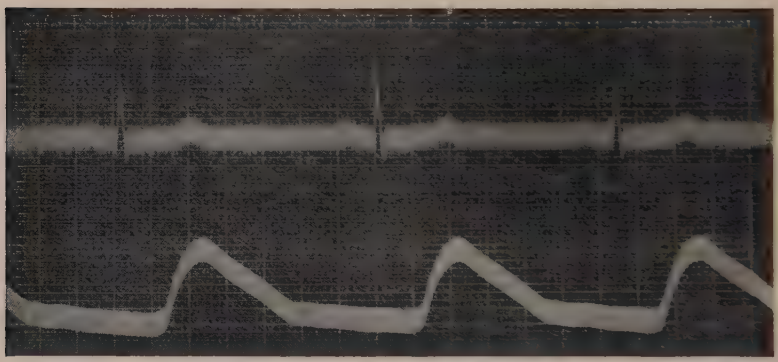


FIG. 37.—"Icteric" bradycardia, the slow pulse of jaundice. Man, age 40, carcinoma of pancreas with extreme jaundice, icteric index 84. Pulse rate about 46.

SINUS ARRHYTHMIA

No irregularity of the heart rhythm has been the subject of more scientific speculation than that of sinus arrhythmia or respiratory arrhythmia. This phenomenon is recognized in all mammals, and is particularly well seen in dogs and young children. It consists of a gradual speeding up of the pulse rate with inspiration and a slowing on expiration. It has been aptly compared to *crescendo* and *diminuendo* as used in harmonics. Analysis of such respiratory arrhythmias shows an orderly sequential time relation, where each diastolic period either becomes larger and larger, or smaller and smaller.

At times the abrupt change at the end of inspiration or expiration may give such a pronounced arrhythmia, that the condition appears to the examining physician almost pathologic.

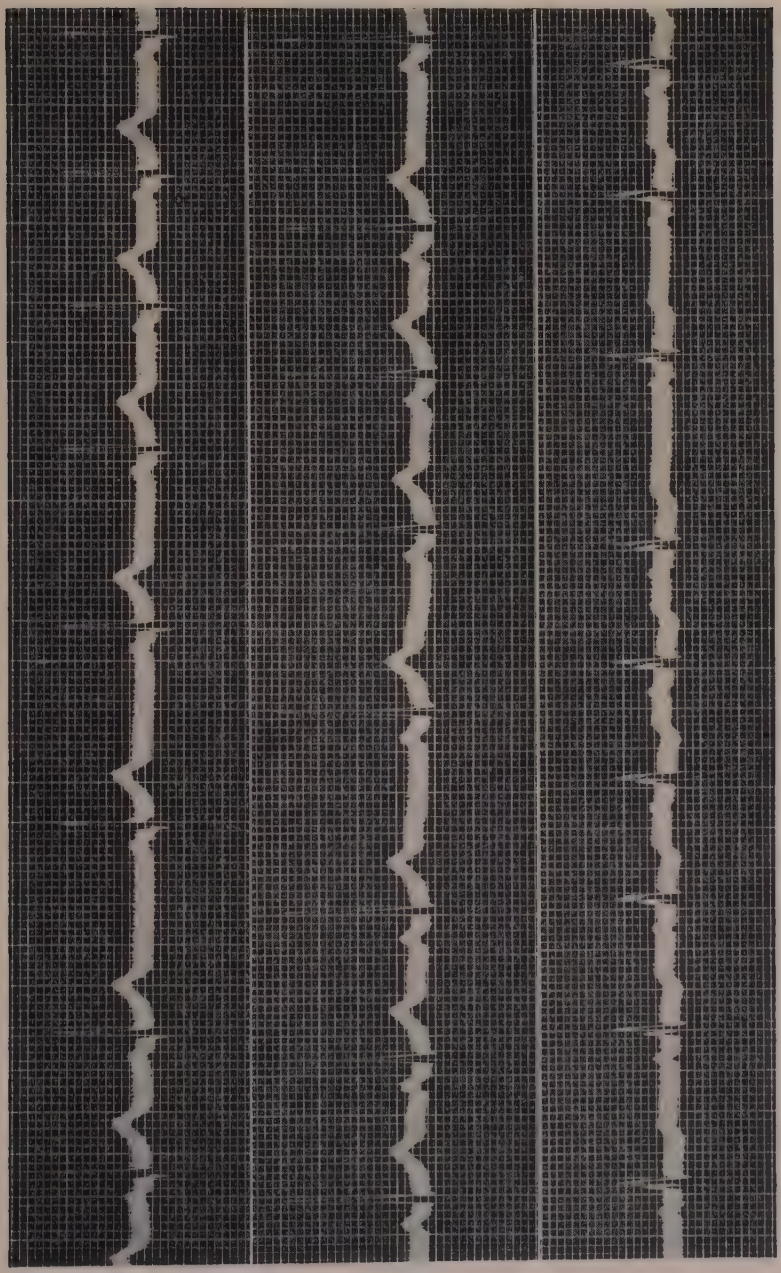


Fig. 38.—Normal sinus arrhythmia. Boy, age 11.

FIG. 39.—Extreme sinus arrhythmia. Girl, age 11. Note the abrupt change in rhythm.

The condition has no special significance, but unless studied carefully, may be mistaken for other irregularities of the heart beat.

PREMATURE SINUS NODAL BEATS

The orderly metabolic process governing the pacemaker mechanism may, as a result of intrinsic cell disturbance, release an impulse prematurely. Such a condition will be noted clinically as an *extrasystole*. Such extrasystoles are rather uncommon. We have noted but four in more than 6,000 records.

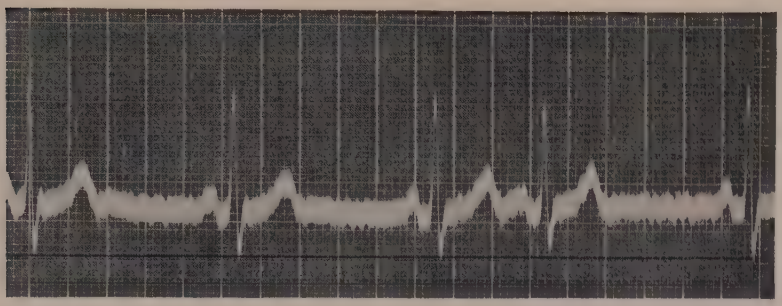


FIG. 40.—Sinus nodal extrasystoles. Man, age 51. Note that the extrasystoles in all respects resemble normal cardiac cycles. The size, configuration, and the elements are the same as in the normal complex. The premature beat is not compensated.

Of special interest is the fact that premature sinus nodal extrasystoles are not compensated, in contradistinction to all other types, except those that are interpolated.

PACEMAKER DELAY OR BLOCKED SINUS NODAL IMPULSES

No condition jeopardizes the life of an individual more, than failure of the pacemaker to develop an impulse for the cardiac cycle. Blocking of the impulse and prevention of its spread through the auricles may result in complete

cardiac standstill and instantaneous death. Many hitherto unexplainable sudden deaths, especially in children, may have been due to such a pacemaker disturbance.

The hazards of such possibilities have not gone unrecognized by nature in the development of the heart. The auriculo-ventricular node, as has been previously described in Chapter I, has a histologic structure similar in many respects to that of the sino-auricular node. Its cells also have the peculiar selective function of being able to develop electric stimuli for the contraction of the ventricles. Its metabolic rate, however, is much slower than that of the sinus node. The auriculo-ventricular node is therefore, in all respects, equipped to become the pacemaker of the heart whenever called upon. In the normal heart, with the upper pacemaker generating its stimulus at a rate of 72 times per minute, the lower pacemaker never has an opportunity to spontaneously develop its own impulse, for it is continually subordinate to the faster stimuli coming down from above. When, however, the upper pacemaker fails to release an impulse, the auriculo-ventricular node now assumes the rôle of the pacemaker of the heart and it liberates its impulse, so that both the auricles and ventricles contract at the same time. The impulse spreads backwards into the auricles, taking the usual pathway in a reverse manner. The stimulus travels down the bundle system to the ventricles in the usual way, so that the ventricles now contract.

This lifesaving mechanism on the part of the lower pacemaker is also exhibited in cases of complete auricular and ventricular dissociation. In this latter condition, both pacemakers are working; the upper one controlling the contraction of the auricles, and the lower one that of the ventricles. This condition will, however, be discussed more completely in the chapter devoted to heart block.

Inasmuch as the intrinsic stimulus production rate of the upper node is more than twice as fast as the lower, two stimuli arising in the sinus node may be blocked before the

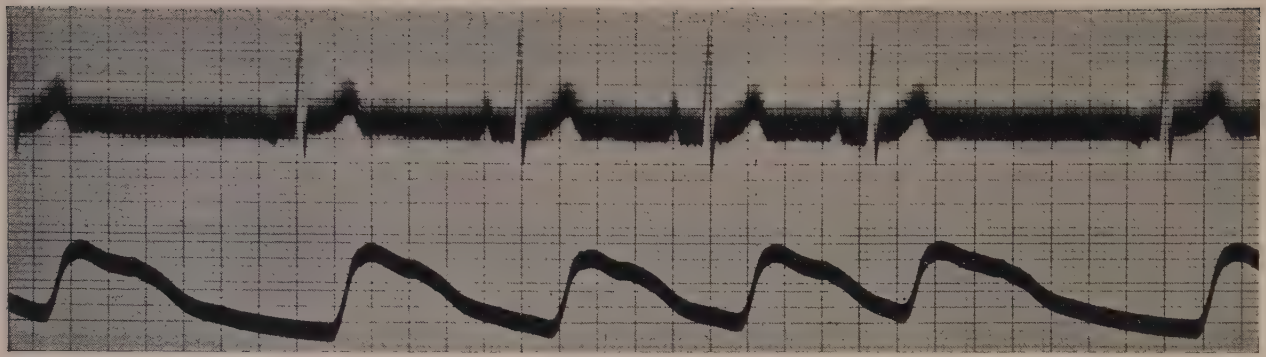


FIG. 41.—Sinus nodal block with another portion of the auricle assuming the rôle of the pacemaker. Note that the impulse arising at an ectopic focus must assume a new pathway through the auricle. The P wave is thus directed downward. The P-R interval of this ectopic beat measures 0.12 seconds, whereas the normal P-R interval measures 0.20 seconds. The radial pulse has been taken simultaneously with the irregularity in order to demonstrate how deceptive palpation of the radial pulse alone may be under such conditions. The impression is given that the irregularity may be due to a premature beat. This record is of special value in pointing out the difference between premature beats, extrasystoles, and ectopic beats. As mentioned in the text, these terms have been used interchangeably as being synonymous with each other. In this case the irregularity is an *ectopic beat* in that it does not arise in the normal pacemaker. *It is not, however, premature;* in fact, the beat is considerably delayed.

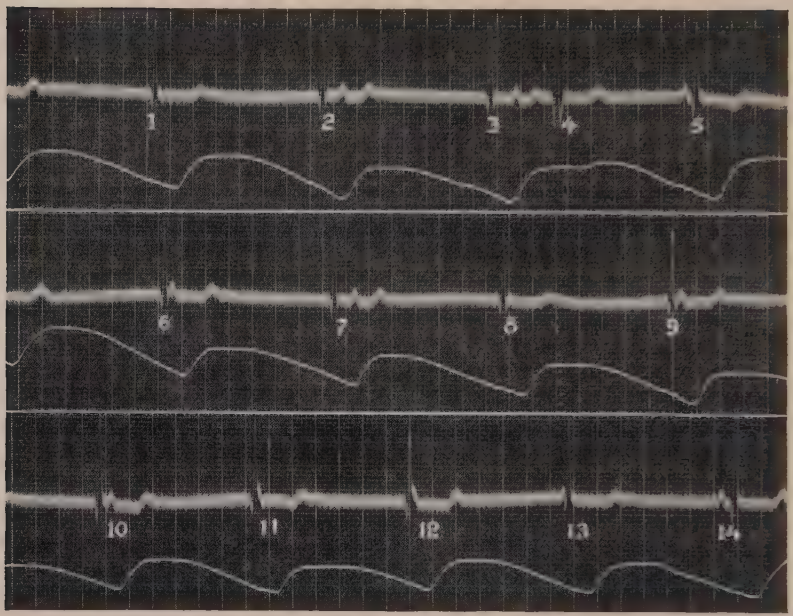


FIG. 42.—Alternation of the pacemakers. Woman, age 56, subjective complaint—heart consciousness. Note that the relations of the P and QRS complexes are constantly changing. The P waves are occurring at a more or less regular rate of from 36 to 44 beats per minute. The QRS complexes, however, are occurring at a slightly more rapid rate. In other words, the lower pacemaker is developing its stimuli for contraction at a slightly faster rate than the upper pacemaker. The result is that the ventricles develop an independent rhythm whenever a stimulus fails to reach the node of Tawara before the latter releases its own impulse. When, however, the impulse from above reaches the lower node before the latter has expressed itself, a normal cardiac cycle occurs. See beat no. 4. When the lower node assumes the rôle of the pacemaker, the P wave is found occurring either at the same time or after the QRS complex. For example, in beat no. 13 the P wave is found coalescing with the upstroke of the QRS complex; in beat no. 3 it coincides with the QRS complex. In beat no. 12 it is found in the descending limb of the QRS complex, in beat no. 2 it is found between the QRS complex and its T wave, in beat no. 4 it is found after the T wave, and now, inasmuch as it occurs before the lower node can exhibit its activity there is called forth a normal cardiac cycle. It is interesting to point out that this normal beat resembles in many respects a premature beat and it is not felt at the radial pulse. Thus the paradoxical situation occurs that because the pacemaker has become diseased and is unable to release its impulse at its usual frequency, the lower node becomes the pacemaker of the heart with a more or less regular pulse. When the upper pacemaker becomes effective the resultant "normal beat" is felt as an extrasystole.

This rare condition of alternation of the pacemakers must be sharply differentiated from third degree heart block or complete auricular and ventricular dissociation. In this latter condition both the auricles and the

lower node becomes active. We thus may have, one, two, or three sinus nodal stimuli blocked, or any ratio of this condition.

Very rarely, some other part of the auricles may temporarily assume the rôle of the pacemaker when the sinus node fails to release an impulse. The mechanism of this phenomenon is not quite clear; various theories have been propounded to explain its occurrence, the most plausible is that of Winterberg, who suggests that certain cells within the auricle may embryologically have retained some of the functions of the sinus nodal cells, and that because of their small number or unfavorable position, never have an opportunity to express themselves. When, however, the sinus node fails to send an impulse, which ordinarily in sweeping through the auricular tissue would discharge the latter nascent impulse, it now has an opportunity to express itself by releasing its impulse. Such an impulse, because of its ectopic focus, must assume a different configuration in the electrocardiographic tracing. It should be inverted and have a very short P-R interval.

When the pacemaker has been so long in developing its impulse that the lower node assumes the rôle of stimulus production, there may be an interplay of the two pacemakers. First, the sinus node generates its impulse and then the lower node expresses itself. When such a condition occurs, the phenomenon of *alternation of pacemakers* develops. It may be diagnosed from the electrocardiographic tracings by noting the constantly changing position of the P wave; at times it may lie a few hundredths of a second before the QRS complex, or may be imbedded in the upper or lower limbs of the complex; or it may follow the complex

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ventricles are contracting from stimuli released by their respective pacemakers. The auricular rate is normally about 72, while the ventricular rate is about 35. (See tracing no. 86.) In complete heart block no impulses can pass through from the auricles to the ventricles, while in alternation of the pacemaker there is no disturbance of conduction, and whenever an impulse is received from the upper pacemaker in time, the ventricle contracts in response.

and lie between it and the T wave, or it may be combined with the T wave, or immediately follow it.

While these conditions are not especially common, their appearance in the electrocardiographic tracing is readily discernible because of the disturbed normal relationship, and the diagnosis is not difficult.

CHAPTER IV

EXTRASYSTOLES

EXTRASYSTOLES

No irregularity of the cardiac cycle occurs with greater frequency than that of the extrasystolic arrhythmias. Its frequency among the various groups of irregularities has been placed as high as 60 per cent by some authors, and as low as 45 per cent by others. There is no question, however, but that the most frequent irregularity of the heart beat and of the pulse seen by the physician, is due to the phenomenon of extrasystolic rhythm.

If we return once again, to the thought that the highly specialized parts of the mammalian heart still retain certain of their embryological elements of function, the theory of the extrasystolic arrhythmias becomes more simple. Each muscle fibre of the myocardium contains within itself the inherent possibilities of irritability and stimulus production, and while it is true that specialized portions of the heart, like the nodal tissues, have assumed the entire function of stimulus production for the heart, at the same time, under conditions later to be defined any portion of the heart may become the point from which a stimulus can arise.

Theoretically, therefore, any point within the muscle tissue, the conducting tissue, or the nodal tissues, may become temporarily more irritable, and thus, capable of faster stimulus production than the normal pacemaker of the heart. Certain physiological factors concerning rate of impulse production through the various types of tissues found in the entire heart, the phenomenon of the refractory period in heart muscle, and the rate of impulse release, limit to a great extent the myriads of possible points which may become potential pacemakers.

From an experimental point of view, the phenomenon of the extrasystolic changes of cardiac rhythm have been extensively studied; Rothberger in the exposed human heart demonstrated that the following points could be mechanically and electrically irritated and become the focus of

stimulus production:— the area near the sinus venosus in the right auricle, any point on the auricular muscle, the junctional tissues, the base of the right and left ventricles, and the apex of the heart. The configuration given to the electrodynamic change in tracings is striking and in most instances readily discovered by a mere glance at the record.

Extrasystoles are *ectopic beats*, because of their origin in other than the customary pacemaker, and because the course which is followed by the impulse is different than the usual pathway, they present a greater display of electrodynamic activity. This is especially true the further the ectopic focus is removed from the normal pacemaker. If this statement is kept in mind, it can be readily deduced that nodal and auricular extrasystoles will more nearly resemble the normal cardiac cycle than those arising in the ventricles.

It should be stated at this time that the common impression of including extrasystoles, ectopic beats, and premature beats as being synonymous is not always true; extrasystoles *are* ectopic beats, but they may not always be premature. The custom of using premature beats and extrasystoles interchangeably has caused great confusion in certain types of arrhythmia, where the ectopic focus is not only not premature, but actually delayed. We shall have occasion to discuss this condition more fully at a later time.

Within recent times several classifications of extrasystoles have been developed; the oldest of these is that based upon the probable anatomic origin of the ectopic focus. Under this classification, extrasystoles are divided into five groups:

1. Sinus nodal.
2. Auricular.
3. Atrio-ventricular or junctional.
4. Nodal (Tawara nodal).
5. Ventricular
    a. Right ventricular
    b. Left ventricular

Examples of each of these various types of extrasystoles are presented and discussed under the chapters considering the diseases of their special points of origin and, for simplicity's sake, the reader is referred to these special sections.

A second classification of extrasystoles disregards entirely the anatomical origin of the ectopic focus and considers only the *functional aspect* of the extrasystoles in relation to the circulation. From this point of view, all extrasystoles, regardless of their source are divided into two groups:

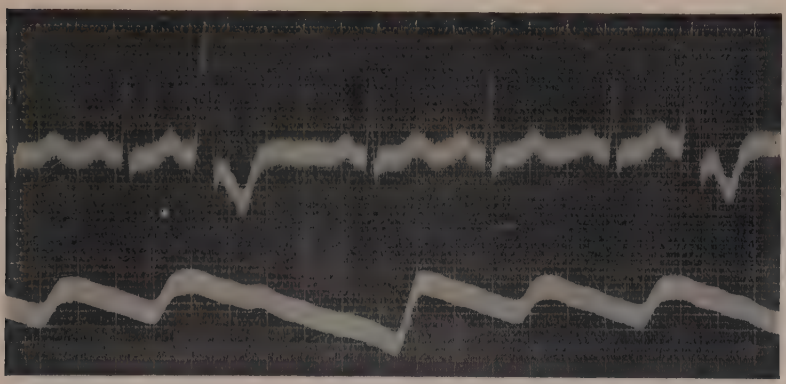

FIG. 43.—Ineffective or exhaustive right ventricular extrasystoles. Note that the extrasystoles occur so prematurely that when the left ventricle contracts there is insufficient blood within it to open the aortic valves and add the impulse to the circulation. The radial pulse shows an intermission at the time that the ventricles are contracting.

first, those which occur so prematurely that the ventricles are forced to contract before they have a sufficient quantity of blood in them to open the aortic valves. When this occurs there has been a cardiac cycle which has failed to add anything to the functional economy of the circulation. In other words, the heart has contracted but no pulsation is felt at the radial artery. Such an extrasystole has been called *ineffectual,* in that it has not effected a change in the peripheral circulation of the individual. If such ineffectual beats occur very frequently it will be seen that the heart is making many useless contractions and is ex-

hausting itself purposelessly. The term—*exhaustive extrasystole* has been used by Wolffe of Philadelphia to describe this condition. Whether or not an extrasystole has opened the aortic valves can be determined clinically by listening to the apical sounds and palpating the radial pulse at the same time. The extrasystole produces three or four heart sounds, while there is no corresponding radial pulse. In fact, an intermission will be felt at the wrist as a result of the *compensatory pause*.

This phenomenon can be demonstrated rather beautifully by the simultaneous photographing of the electro-

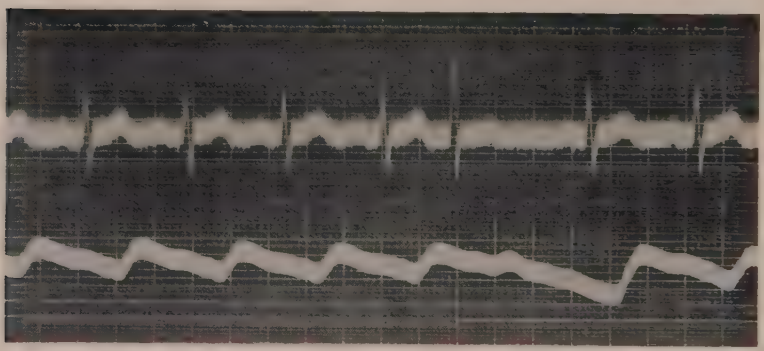

FIG. 44.—Ineffective auricular extrasystoles.

cardiographic and polygraphic tracings and we believe that the true story of the extrasystole can only be determined by this method; examination by the electrocardiograph alone gives no information in regard to the effectiveness of the extra beat, as the degree of prematurity can not be regarded as an index of ventricular filling.

When, however, the extrasystole occurs late enough in the diastolic period to permit the ventricles to be filled with a sufficient quantity of blood to open the aortic valves when contraction occurs, such an extrasystole is spoken of as being *effectual* in that it sends a pulsation to the peripheral circulation. Under these conditions, the heart has made a

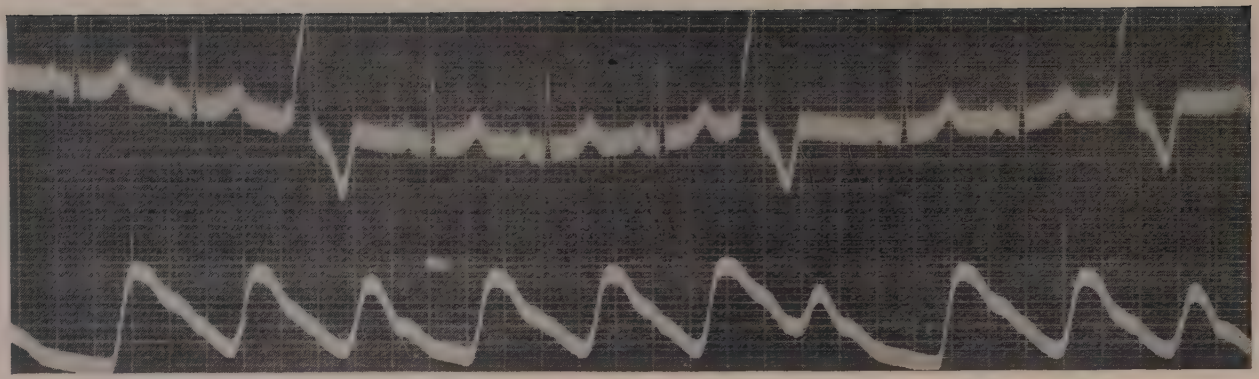

FIG. 45.—Effective or non-exhaustive right ventricular extrasystoles. Note that the extrasystole occurs late enough in diastole to permit the left ventricle to be more or less completely filled. When the ventricle contracts, this volume of blood is sufficient to give an impulse to the circulation and it can be noted at the radial artery. This record is also interesting in pointing out the lack of relation between the electrodynamic phase of the cardiac cycle and the hemodynamic phase. The electrodynamic phase of the extrasystole is represented by huge manifestations of electric energy far greater than that displayed by the normal beat. On the other hand, the hemodynamic phase of the extrasystole expressing itself in the actual work performed by the ventricles is relatively small compared to the normal beat.

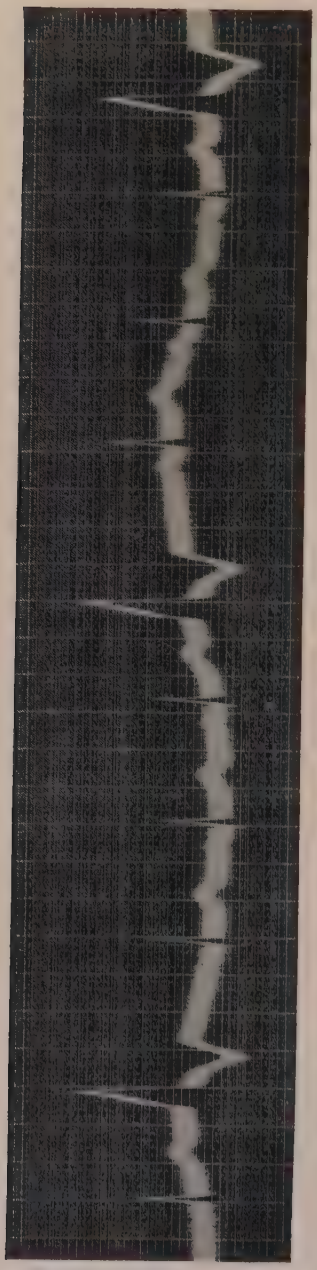

FIG. 46.—Right ventricular extrasystoles due to extracardiac causes. This tracing is from a man, aged 38, complaining of gastric distress. Under the X-ray a very large gas bubble was seen in the stomach; this bubble had elevated the diaphragm and was forcing the heart upwards into the chest cavity. Whenever the patient took a deep breath and permitted the diaphragm to fall, the heart would be relieved from pressure effects and no disturbance of rhythm occurred. When, however, the patient exhaled the rise of the diaphragm would initiate a series of extrasystoles. Simultaneous tracings of his electrocardiogram, radial pulse, and respirations, showed that he developed an extrasystole at every fourth or fifth beat, usually at the time of deepest expiration.

premature beat but nothing is lost to the economy of the circulation as the compensatory pause following the ectopic beat restores the total number of pulsations to normal over a given period of time. In other words, while it is true that the ectopic beat has caused the heart to contract before its usual normal time, the circulation has not suffered as a result of this disturbance of rhythm. Such premature beats are thus known as *non-exhaustive extrasystoles.*

A third classification, disregarding entirely the anatomic origin of the ectopic beat and also the hemodynamic classification, considers only the etiological condition responsible

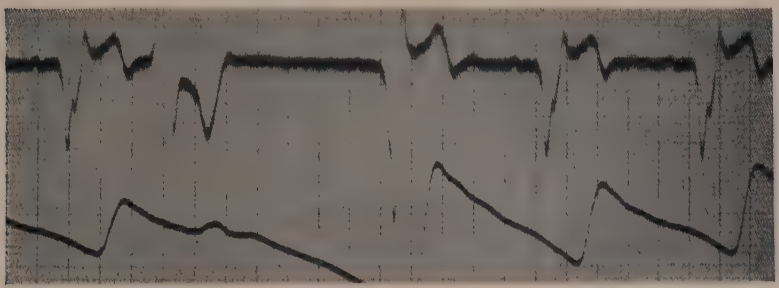

Fig. 47.—Right ventricular extrasystoles occurring in a case of mushroom poisoning. An example of extrasystolic arrhythmia due to ingestion of toxic substances.

for the development of extrasystoles. The underlying factors governing the development of an ectopic focus is considered from this angle to be of the greatest importance, so far as the significance of the extrasystole is concerned. For example, where the exciting factors are due to relatively innocuous causes like extracardiac pressure from an inflated stomach or colon, the extrasystolic arrhythmia may be entirely disregarded, for such cases have been known to persist for more than 40 years without any other disturbance of the heart. Certain toxic substances like nicotine, alcohol, and over-indulgence in caffein-containing beverages, may also be responsible for extrasystoles which promptly disappear upon the removal of the irritant.

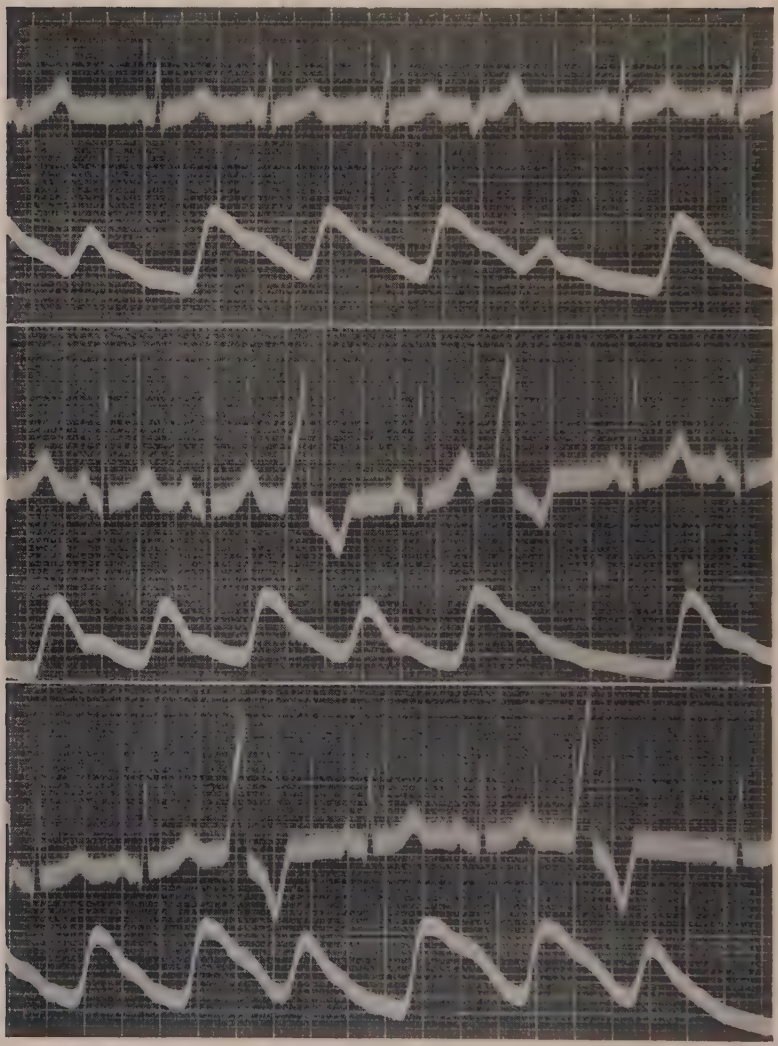

FIG. 48.—Right ventricular extrasystoles occurring during an attack of diphtheria. Boy, age 8. An example of extrasystolic arrhythmia occurring in the course of the acute infectious diseases.

Of more serious significance are those extrasystoles occurring during the course of the acute infectious diseases, such as pneumonia, diphtheria, rheumatic fever, and influ-

enza. The development of ectopic foci in these conditions is a definite expression of myocardial damage and resultant irritability. In addition to the extrasystolic arrhythmia such cases will also show in a majority of instances other

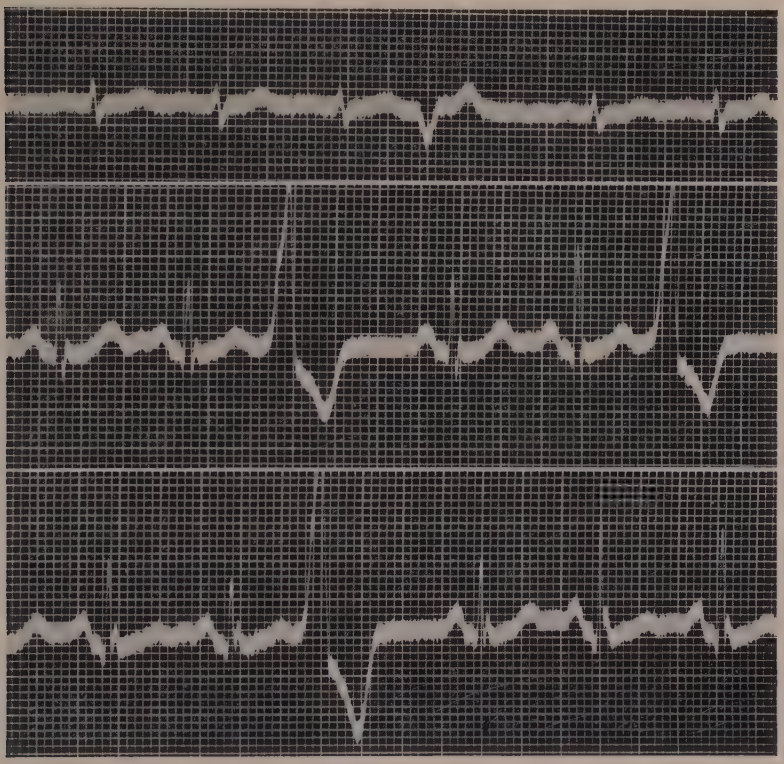

Fig. 49.—Incidental right ventricular extrasystoles. Note here the absence of any other change in the tracing. Girl, age 17, with a negative cardiovascular study, but with history of chronic constipation.

signs of myocardial toxicity; such for example, as prolongation of the P-R interval, delays in the QRS complex, and alteration in the T wave. The occurrence of extrasystoles with such associated electrocardiographic change must be regarded as evidence of serious toxemic myocardial invasion.

A further and probably most serious significance of extrasystoles, is that associated with the coronary artery syndrome. Where there has been clinical, historical, and instru-

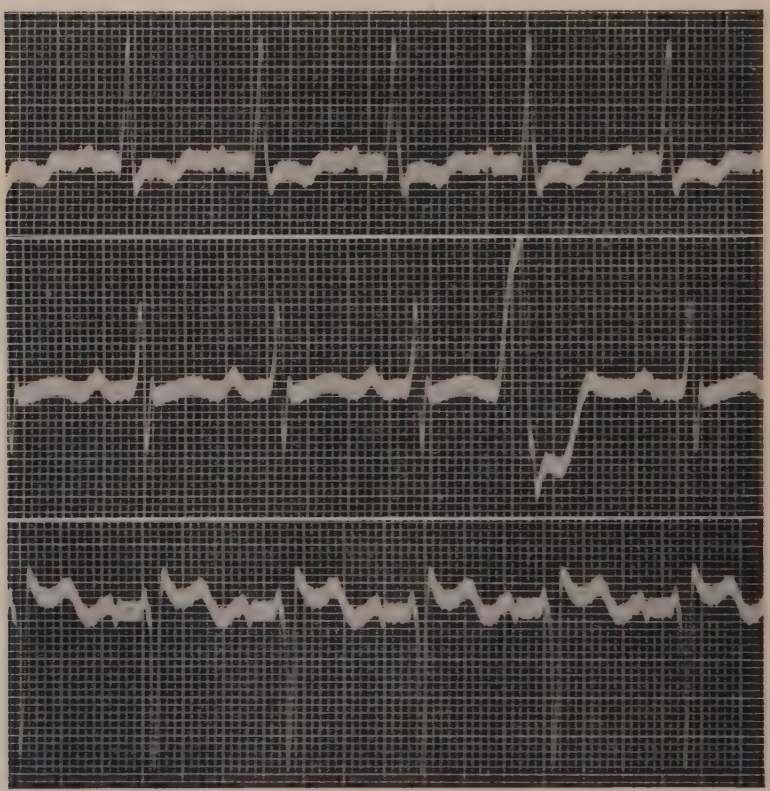

FIG. 50.—Fundamental extrasystoles occurring in a man, age 53, with a positive Wassermann test, hypertension, aortic insufficiency and aortitis. In this tracing the right ventricular extrasystole resembles that seen in Fig. 46. Its significance must be considered in relation to the other evidences of serious myocardial disease seen in the tracing. For example, the P wave is split, the P-R interval is lengthened to 0.26 seconds, the QRS complex is delayed to 0.10 seconds, there is a well marked left axial deviation of the heart, and the T waves in Lead I are bowed and inverted.

mental evidence of coronary artery sclerosis, the development of extrasystoles is to be regarded as ominous: many times the development of such beats indicates that the

ectopic focus arises in an area of infarction, and is an expression of focal irritability developing in the necrosing heart muscle. In our experience, the development of an extrasystolic arrhythmia under such conditions has usually been the sign of grave prognosis; more than 95 per cent of such cases having died within the ten day period following the discovery of the ectopic beats.

To all of these classifications, each of which serves a specific purpose, we have attempted to add another, which in a general way serves to combine more than one feature as

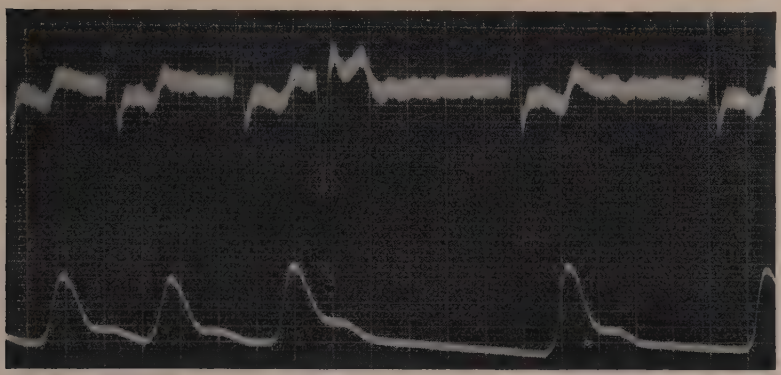

Fig. 51.—Fundamental left ventricular extrasystoles. Woman, age 56, chronic myocardial failure with edema. Note in this tracing the associated pathology, auricular fibrillation, bowed and inverted T waves in Lead II, and the ineffectiveness of the extrasystoles.

the basis of our grouping. We have divided all extrasystoles into two major types: first, those which we have designated INCIDENTAL, comprising all extrasystoles seen in otherwise normal hearts and occurring in otherwise normal electrocardiographic tracings. To these incidental extrasystoles we have attached very little importance except that we regard their occurrence as suggestive of a more or less unstable cardiovascular system. We have found that such incidental extrasystoles are seen most frequently in those individuals exhibiting the so-called vagotonic syndrome. In this syndrome can be included those individuals who show excessive

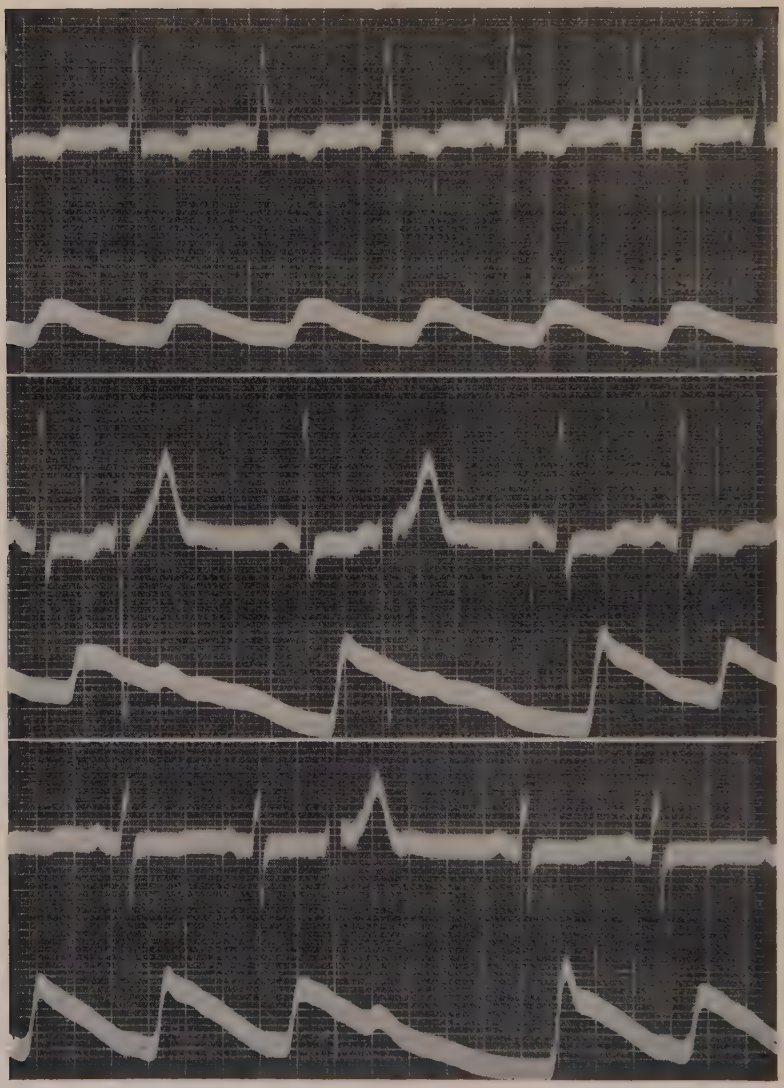

FIG. 52.—Left ventricular extrasystoles of the fundamental type. Note the associated pathology, alteration of the T waves in all leads, and the splitting of the QRS complexes.

reactions to emotional and psychic changes, who have various types of vasomotor instability, and who react to mechanical and toxic stimuli excessively.

Into our second group, classified by us as FUNDAMENTAL, we have included all extrasystoles found in conjunction, or associated with other evidences of myocardial mischief, either in its acute or chronic stage. Such, for example, would be the extrasystoles found during the severe toxemias of acute infections, and also in coronary artery sclerosis and infarction.

In using the terms *incidental* and *fundamental,* we have attempted to call attention to the possible significance of the extrasystoles. Incidental extrasystoles are found only in otherwise normal tracings, whereas fundamental extrasystoles are only seen in tracings exhibiting other signs of cardiac pathology.

While each classification of the extrasystolic arrhythmias serves a definite specific function, we believe that the clinician will obtain more information in using the descriptive terms incidental and fundamental, than in any other form of grouping.

INTERPOLATED EXTRASYSTOLES

It has been previously pointed out that all extrasystoles with the exception of those arising at the pacemaker are compensated; the refractory period of the heart muscle being responsible for this phenomenon. Very rarely an extrasystole may arise at a midpoint in diastole without affecting the dominant rhythm of the heart. Such extrasystoles are known as *interpolated extrasystoles.* If the rate of the heart beat is relatively slow, this interpolated beat may be felt as a smaller pulsation between two normal beats at the radial artery. Sometimes, however, this smaller beat cannot be palpated and it passes unrecognized. No special clinical significance can be attached to interpolated

extrasystoles whether they be effective or not; they are of significance only in so far as they may be considered as incidental or fundamental.

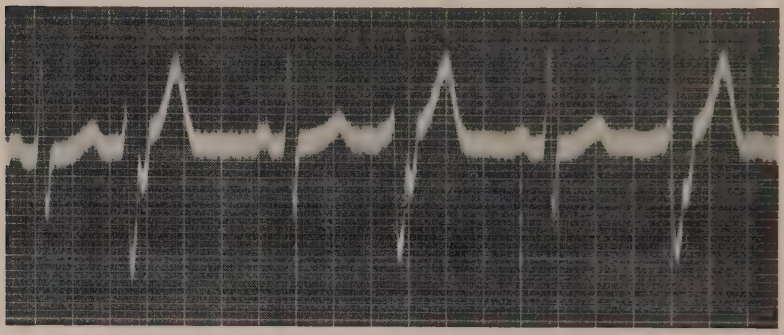

Fig. 53.—Interpolated left ventricular extrasystoles.

PAROXYSMAL TACHYCARDIA

No irregularity of the heart beat has been more misunderstood than that of the *Paroxysmal Tachycardias*. As the name implies there is an excessive rate of cardiac contraction which begins and ends abruptly; it is readily distinguished from a sinus tachycardia in that the latter never slows up or speeds up within the space of a single beat. Sinus tachycardia shows a gradual increase or decrease in its dominant rhythm. In marked contrast to this, paroxysmal tachycardia is the development of a new rhythm instantaneously.

Although many theories have been employed to explain the mechanism underlying paroxysmal tachycardia the current belief indicates that the condition is primarily an extrasystolic arrhythmia. Paroxysmal tachycardias, therefore, may be classified according to the origin of the ectopic beat precipitating the new rhythm. Such an ectopic focus is conceived as being exceedingly irritable and liberating a series of stimuli for contraction. The stimuli may be released at a rate of from 180 to 260 per minute.

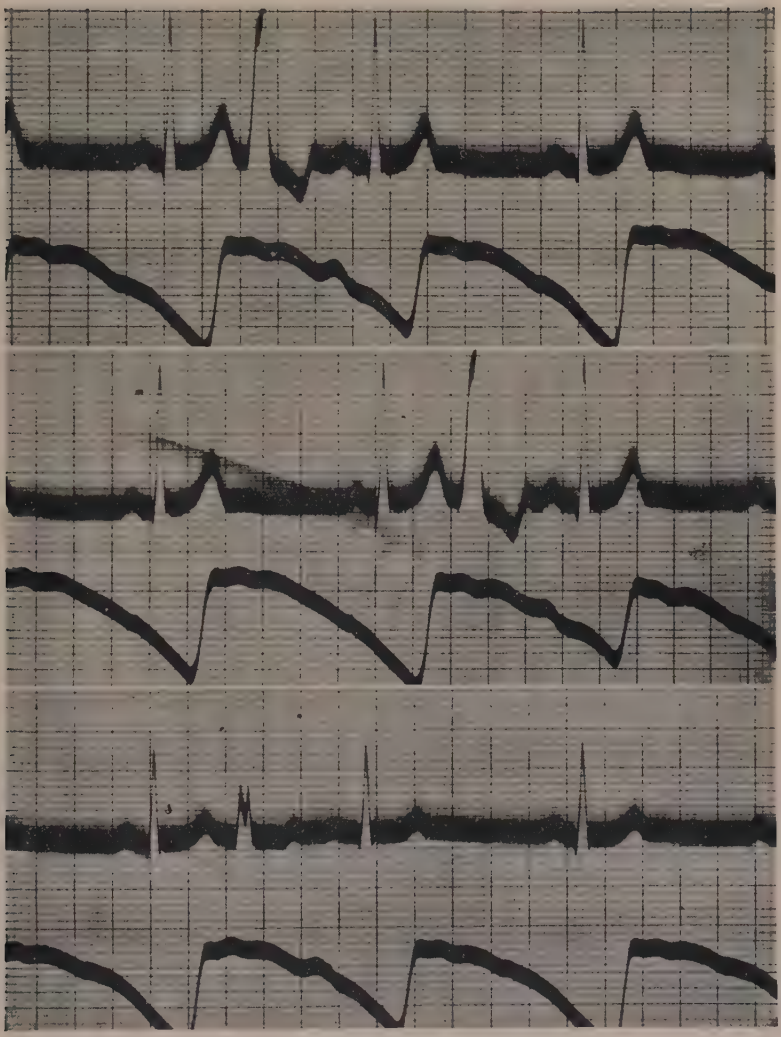

FIG. 54.—Interpolated right ventricular extrasystoles.

In general, there are three possible points which may become the ectopic foci in this new rhythm: the auricles, the atrio-ventricular node, and the ventricles. We may thus have auricular paroxysmal tachycardia, nodal par-

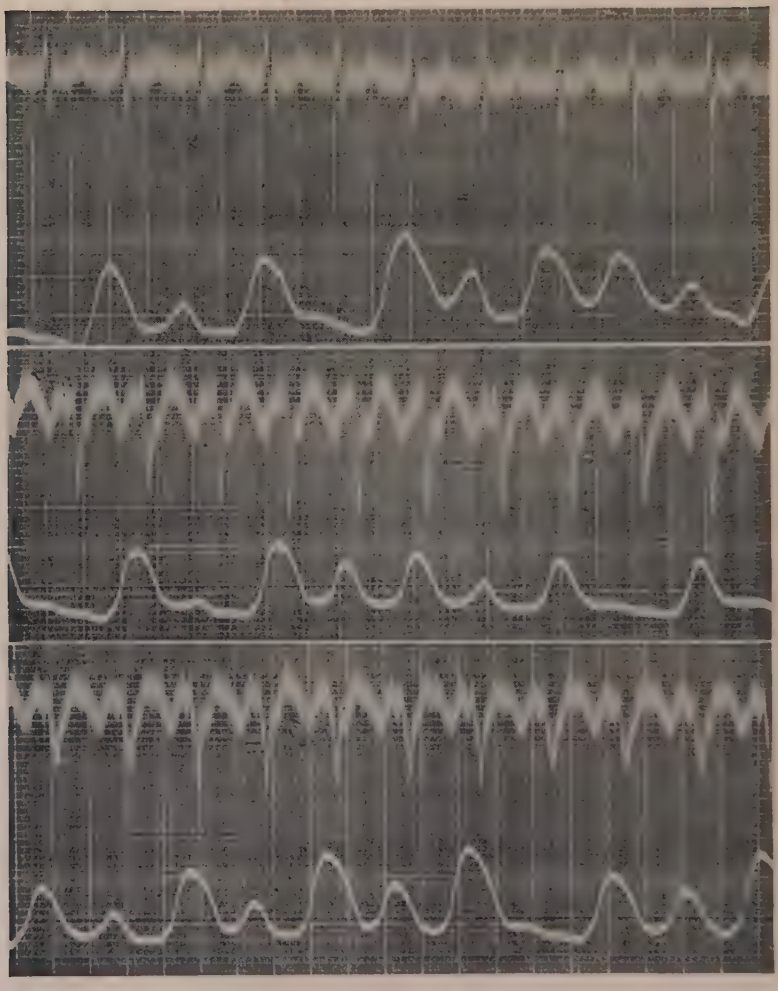

Fig. 55 A.—Paroxysmal auricular tachycardia occurring in a patient during an attack of post-infectious sore throat.

oxysmal tachycardia, and ventricular paroxysmal tachycardia, depending upon the point of origin of the extrasystoles.

It may be very difficult in examining an electrocardiographic tracing to determine which type of paroxysmal

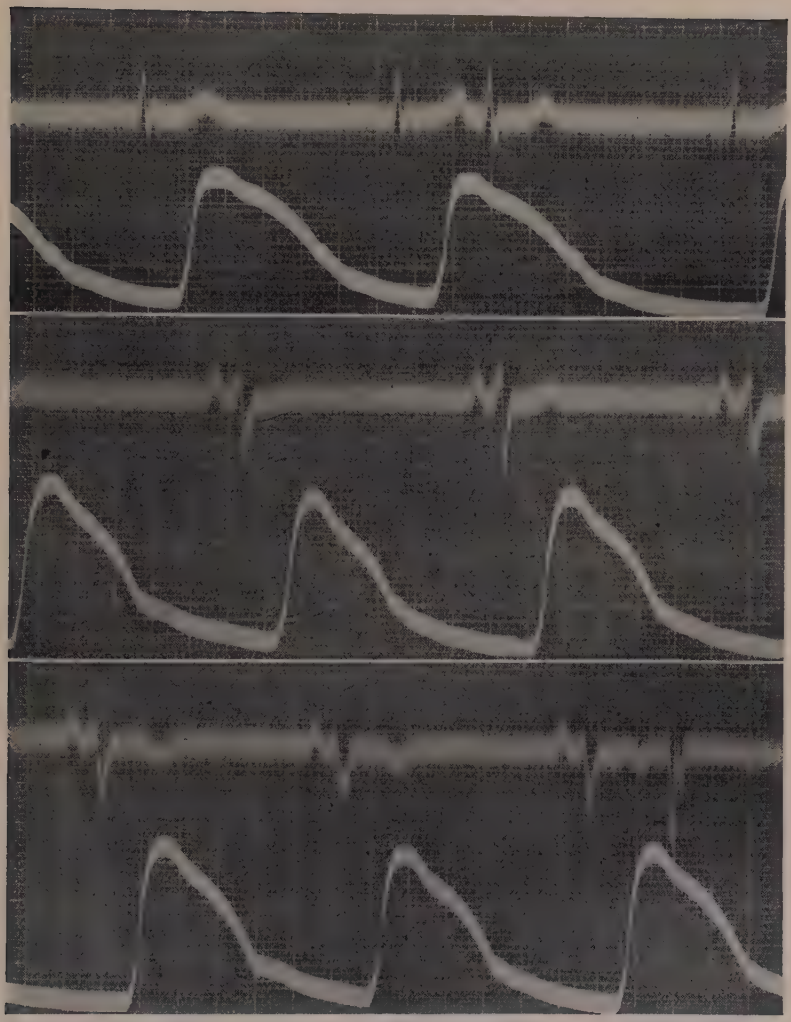

Fig. 55 B.—Paroxysmal auricular tachycardia occurring in a patient during an attack of post-infectious sore throat. Same patient 24 hours later. Note the occurrence of auricular extrasystoles; these demonstrate the origin of the paroxysm.

tachycardia is present; if the beginning and the end of the attack have been recorded, there is usually found a separate or coupled extrasystole in the normal rhythm. The

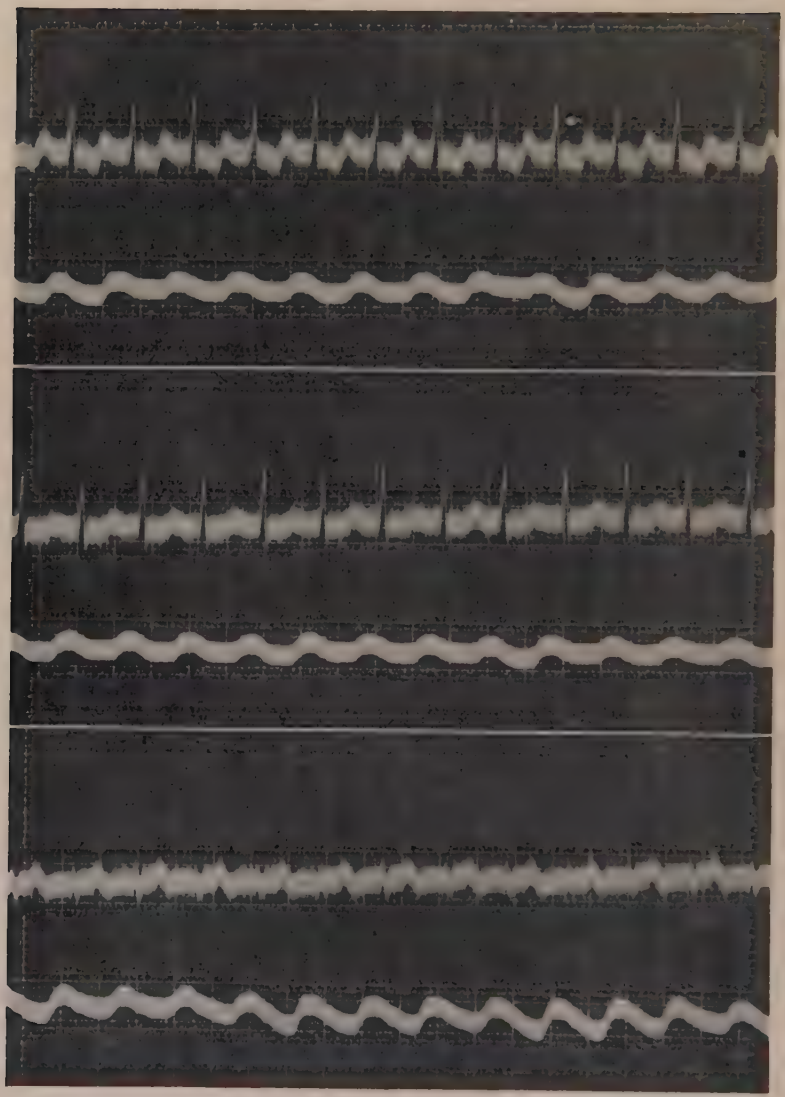

FIG. 56.—Paroxysmal tachycardia. Nodal origin. Rate about 200.

diagnosis of this extrasystole will give the type of tachycardia present.

True paroxysmal tachycardia of extrasystolic origin must

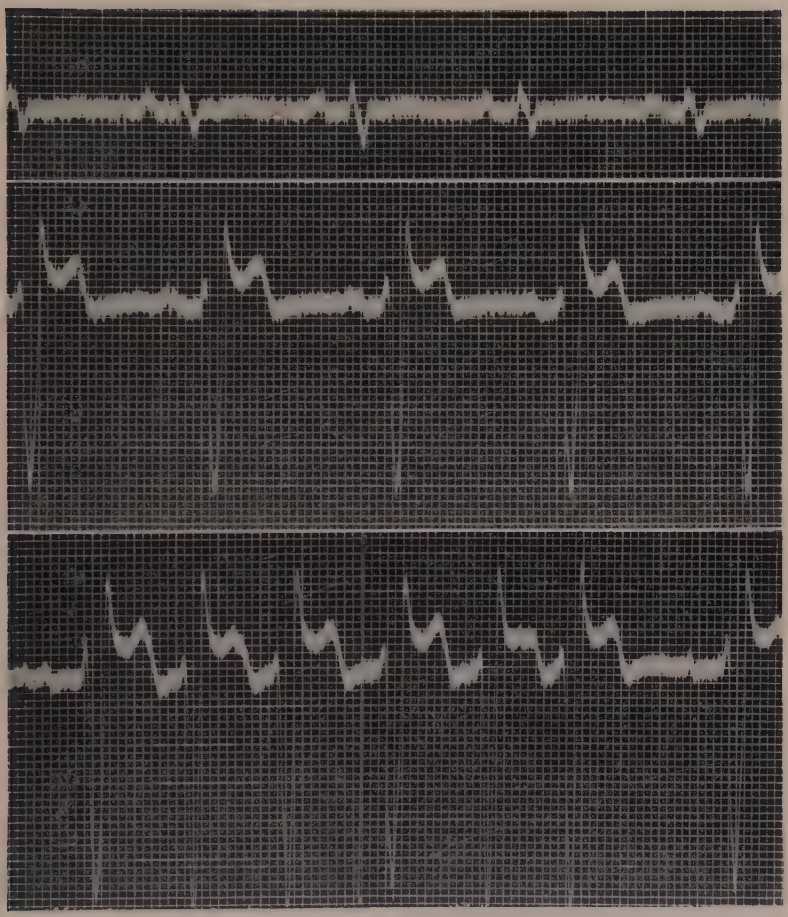

Fig. 57.—Paroxysmal ventricular tachycardia.

be distinguished from the so-called tachycardia of paroxysmal auricular fibrillation or flutter. In such cases the normal rhythm may be interrupted by periods of very rapid auricular fibrillation or flutter. Electrocardiographic studies may reveal the pathology without difficulty, but it may be impossible to make the diagnosis by clinical methods alone.

CHAPTER V

DISEASES OF THE AURICLES

DISEASES OF THE AURICLES

Anatomically and histologically, the right and left auricles of the mammalian heart may be regarded as continuous; except for a more or less complete separation by a thin membranous septum they may be regarded as acting in unison as one chamber. The interauricular septum is perforated in the embryo by a window known as the *foramen ovale.* This connection between the two auricles gradually becomes smaller as the individual reaches adolescence, and it may entirely disappear in certain individuals. In others, however, the interauricular connection may remain permanently open throughout their life and under ordinary conditions might not be of any significance. Where, however, there has been myocardial damage and where there has been unusual stress or strain upon one auricle or the other, the persistence of such an interauricular connection may seriously enhance the pathological progress.

From a clinical point of view, the diseases affecting the auricles are usually the result of ventricular change. A typical example of such a process occurs in the pathogenesis of auricular fibrillation in rheumatic mitral stenosis. In this latter condition, as a result of the narrowed mitral valve, the left auricle dilates and hypertrophies to take care of the added burden placed upon it in forcing the same volume of blood through the constricted valve orifice. The stretching of the auricular muscle, like the stretching of any muscle, causes it to become hyperirritable. This hyperirritability is communicated equally to both the right and left auricles, although the initial process appears to occur in the left auricle first.

The first expression of this hyperirritability appears to be the development of ectopic foci of stimulus production in the auricular muscle. The resultant premature beats or extrasystoles are easily distinguished electrocardiographi-

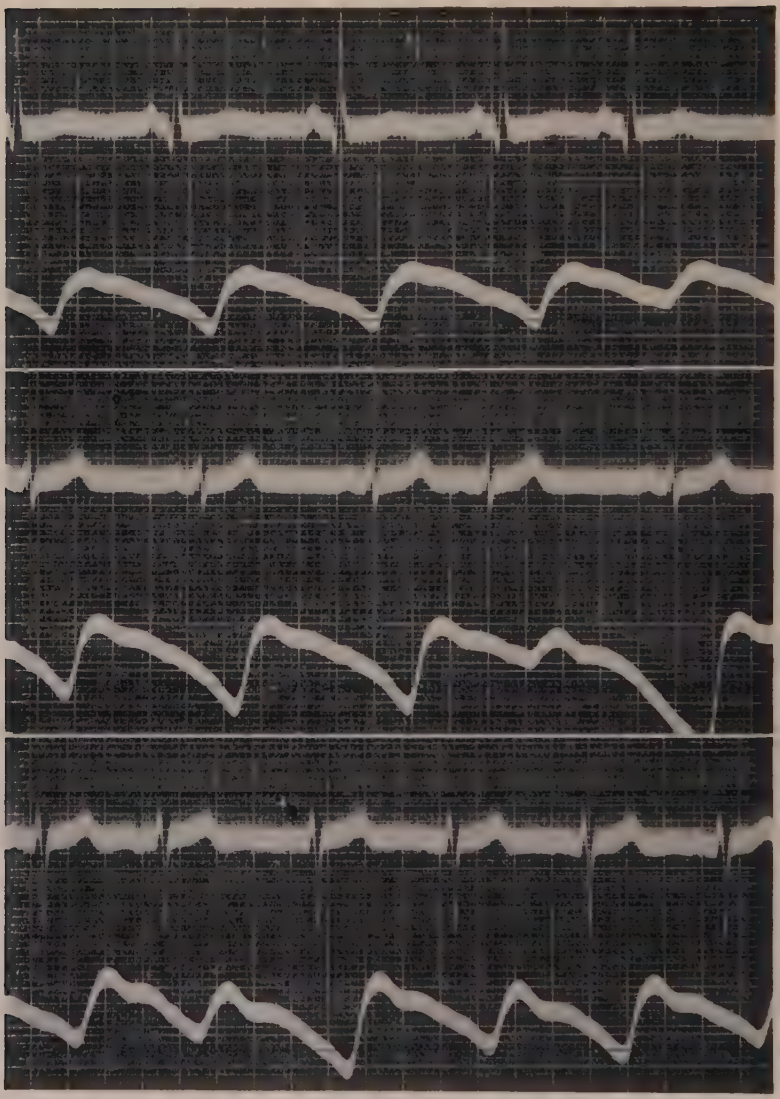

FIG. 58.—Auricular extrasystole arising near the sino-auricular node. Note that the P wave is well formed and upright, and that the entire beat resembles very closely a normal one, the only exception being that the P-R interval is shortened to 0.14 seconds and the beat is premature. It is followed by a compensatory pause.

FIG. 59.—Auricular extrasystoles arising at a point theoretically midway between the sino-auricular node and the atrio-ventricular junction. Note here that the P wave tends to be diphasic and that the P-R interval is shortened to about one half the normal conduction time—0.10 seconds.

cally. While these extrasystoles may arise from any point of the musculature which is hyperirritable, they may be roughly classified into three general groups. First, those arising in points of the auricle near the normal pacemaker; secondly, those arising at a point near the auriculo-ventricular junction; and thirdly, those arising at a point midway between.

Regardless of their point of origin, *auricular extrasystoles* resemble in all respects the normal sinus cardiac cycle; the P wave, however, may be somewhat altered, and the P-R

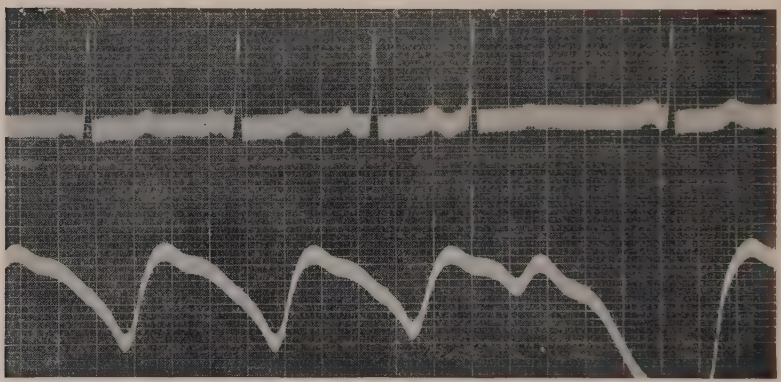

Fig. 60.—Auricular extrasystoles arising near the atrio-ventricular junction. In this case the P wave is inverted and the P-R interval is reduced to about 0.04 seconds.

interval changed. Of the many explanations given for these changes, the most lucid is that of the Wenckebach and Winterberg school. In those auricular extrasystoles which arise near the normal pacemaker, the P wave is usually upright and well formed, and it is distinguished from a normal pacemaker beat only through its prematurity. As the ectopic focus moves further away from the pacemaker toward the auriculo-ventricular junction, the P-R interval may become shortened, because of the smaller pathway traversed by the impulse in order to reach the lower node of Tawara. Inasmuch as the impulse for the auricular contraction now has

to run backward through the auricle, the P wave becomes inverted. If the ectopic foci lie at a theoretical point midway between the sinus node and the auriculo-ventricular node,

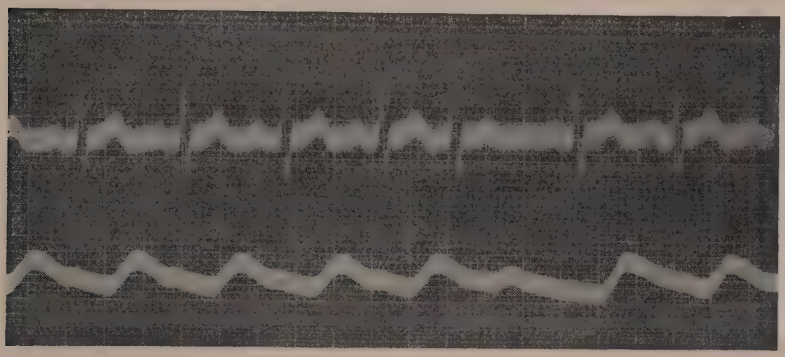

FIG. 61.—"Occult" or hidden auricular extrasystoles. Due to the accelerated heart rate, the T-P interval representing the diastolic period is shortened to such a degree that the P wave of the premature auricular extrasystole lies buried in the preceding T wave. It is discovered only by a careful examination of the T waves.

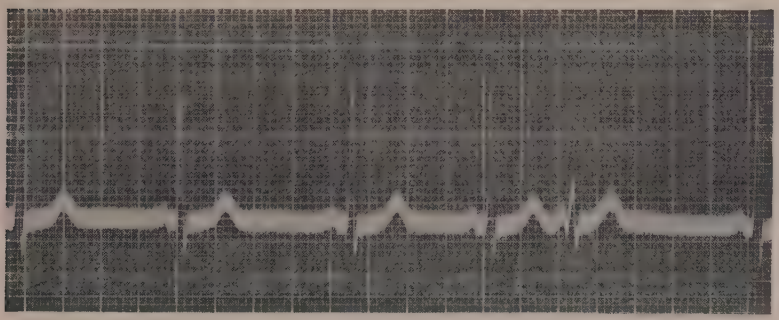

FIG. 62.—Very premature auricular extrasystole. The ventricular complex of this cardiac cycle is somewhat altered in form because the ventricle has not quite recovered from the preceding normal beat.

the P-R interval will be shortened and the P wave may become diphasic.

Frequently it may be impossible to distinguish the characteristics of the P wave, as it may lie buried in the preceding T wave. Under these conditions it may only be dis-

covered by a careful examination of the T waves. Usually the T waves containing the buried P waves are a little larger and of greater amplitude than the other T waves in the tracing.

If the auricular extrasystole is very premature, it may find the bundle tissue somewhat refractory. The ventricular complex which then follows may be slightly different in configuration than others in the series.

AURICULAR FLUTTER

Probably no abnormality of the heart rhythm has been more vigorously discussed than that of auricular flutter. The condition can be briefly described as an exceedingly rapid series of auricular contractions at a rate approaching 300 per minute. At this very rapid rate the ventricles find themselves unable to follow every contraction of the auricles, so that a physiological block develops, and only every second or third stimulus passes through to the ventricles. The ventricular rate may therefore be very rapid and somewhat irregular.

The causation of this very rapid auricular rhythm is a matter of some conjecture. The English school of cardiologists led by Lewis, has attempted to explain the condition by assuming a so-called circus movement, or a wave of electric impulses passing around in a circle at the orifices of the large veins at the sinus venosus. As the impulses swing around this closed circuit, there is released a constant deluge of impulses which spread over the auricles in all directions, causing them to contract at a very high rate.

The German school, however, has conceived the process as being one of a series of auricular extrasystoles occurring from many ectopic foci, but at a rate too fast for the ventricles to follow.

Electrocardiographically, auricular flutter is readily discernible. There is presented a well defined series of P waves

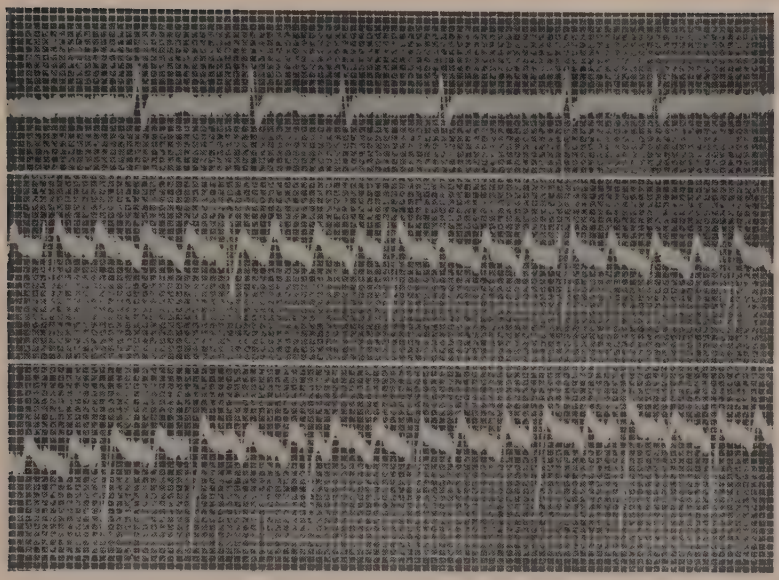

FIG. 63.—"Slow" auricular flutter. Note that the P waves are usually well formed and resemble the normal type. The auricular contraction rate varies from 220 to 240 per minute. Due to physiological block which has developed, the ventricular rate varies from 120 to 140 beats per minute.

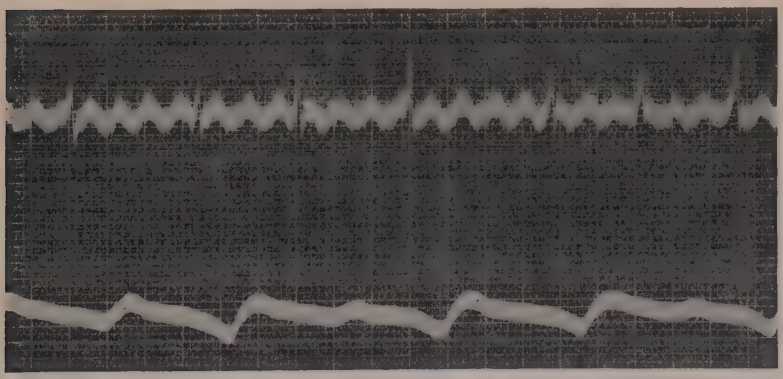

FIG. 64.—"Rapid" auricular flutter. In this case the P waves are well formed but at times two or three waves appear to coalesce with each other. There appear to be about 280 to 300 auricular contractions per minute. The ventricular rate is also very rapid and irregular.

followed somewhat irregularly by ventricular complexes. The P waves coalesce with the T waves of the ventricular complexes, but careful examination of the tracing will disclose this condition. The auricular waves may be poorly defined in certain leads; they are most frequently seen to the best advantage in Leads II and III, but it may be necessary to examine all three leads carefully for a correct diagnosis of the condition.

AURICULAR FIBRILLATION

The phenomenon seen in auricular flutter is accompanied by complete contraction of the auricles; the auricular musculature may rapidly become exhausted and behave like any other muscle of the body at its fatigue point. Instead of contracting as a whole and with coördinated response to each stimulus, the various portions of the muscle become so irritable that only a partial and incomplete response of the contractile elements of the myocardium results. Such a muscle, instead of presenting a regular wave of contraction, shows a quivering movement when examined carefully *in situ*.

The development of this quivering or fibrillation of the muscle fibres is now generally considered to be a secondary stage of auricular flutter. Auricular fibrillation is probably one of the oldest forms of cardiac arrhythmias discussed in the literature. The physicians of a century ago discussed the clinical picture under the very descriptive term "delirium cordis" and "pulsus perpetuus irregularis." The matchless observer Mackenzie demonstrated the true significance of auricular fibrillation; he pointed out that in the rapid incoördinated quiverings of the auricular musculature, stimuli varying both in frequency and in intensity, were sent down to the ventricles. Their response was consequently irregular, both in rate of contraction and volume of blood expelled. In the light of recent physio-

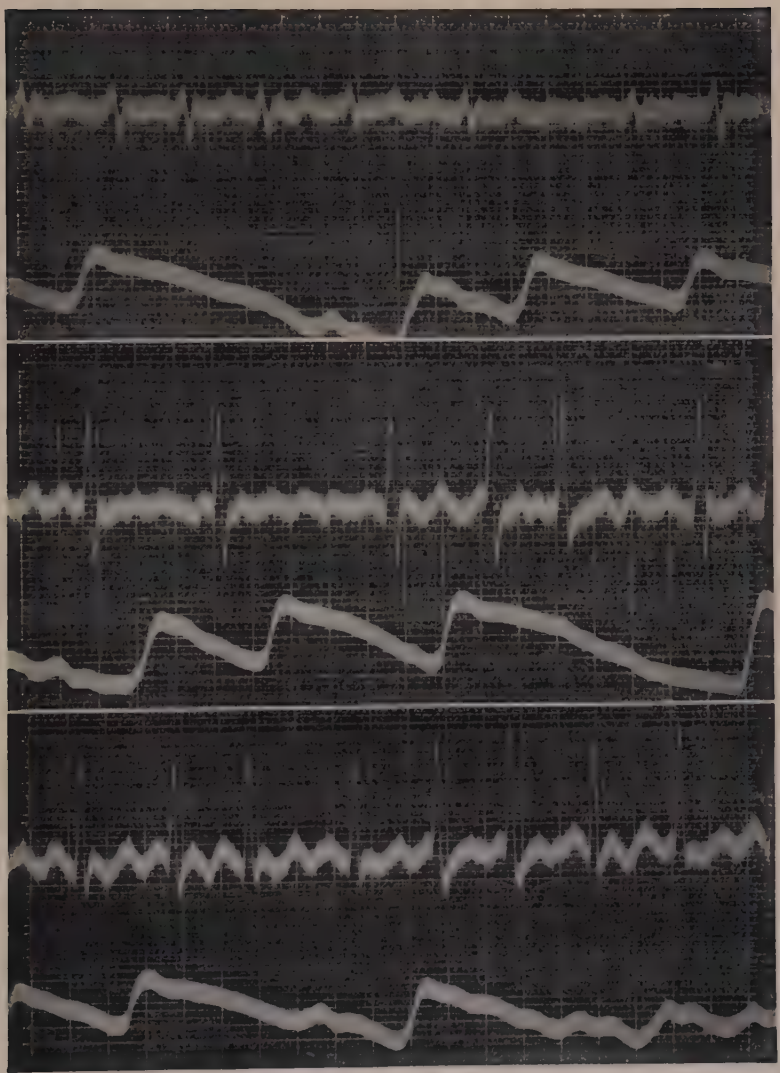

FIG. 65.—Auricular fibrillation with coarse circus movement. The coarse oscillations of the string resemble at times the P waves in auricular flutter; whenever these coarse movements coincide with the T wave of the ventricular complex the latter may assume unusual forms.

logic investigation, it seems probable that the electrodynamic phenomena seen in auricular flutter, may be responsible for the development of fibrillation. According to the circus movement theory, the rate of annular stimulus movement becomes faster and more irregular, and as a result, stimuli are sent out in showers throughout the auricles; these stimuli, bearing no relation to each other, follow different pathways, and the resulting disturbance prevents the various portions of the auricular musculature from contracting in other than a very unregulated fashion. On the other hand, the German theory of ectopic foci tends to explain the condition as being due to myriads of irritable points in the auricular muscle, each of which attempts to develop a stimulus for contraction. Because of their multiplicity, these impulses tend to neutralize each other, so that only the local area is concerned in muscular response. No coördinated contraction can thus occur, and the condition of minutely localized contractions arises.

When studied electrocardiographically, auricular fibrillation presents no difficulties of recognition. In the absence of any coördinated auricular contractions, no P waves will be found. Due also to the irregular reception of stimuli from above, the ventricular complexes will vary in rate and configuration. Of considerable importance in this regard is the fact that irregularities are constantly present. Where the circus movement is very rapid, the electrocardiographic tracing may be very simple to interpret. The absence of P waves, and the irregular, and usually somewhat rapid ventricular complexes distinguish the condition quite readily.

When, however, the circus movement is slower, the stimuli may be recorded as coarse oscillations of the electrocardiographic string; these coarse oscillations resemble in many respects the P waves seen in auricular flutter. When such coarse circus movement oscillations coincide with the

T wave of the ventricular complex, the latter may assume unusual and bizarre forms.

It may be pointed out here that the simultaneous photographing of the radial pulse with the electrocardiographic tracings of the various leads is of inestimable value in indicating the severity of decompensation. No graphic study of auricular fibrillation can be considered as complete, unless the actual mechanical expression of the ventricles is known in addition to the electrodynamic phenomena. From a clinical point of view, it is important to know the rate of ventricular contraction and the rate of effective peripheral circulation. In a normal sinus rhythm, each ventricular complex is accompanied by a pulsation of the radial artery. If, however, the ventricular contraction has been so fast, or so small that it is unable to open the aortic valves and, hence, deliver its volume of blood to the circulation, the ventricular contraction has served no useful function in the economy of the circulation.

Clinically, this disproportion between the ventricular contraction and the reception of blood in the peripheral circulation is known as the *pulse deficit;* as a general rule, the greater the pulse deficit the more serious is the degree of decompensation. Many times, however, the palpating finger on the radial artery may be deceived as to the rhythm and rate of the pulsations. The ear, too, may also be deceived in regard to the sounds heard at the apex of the heart. Simultaneous polygraphic and electrocardiographic studies in such conditions may bring unexpected knowledge to the clinician. This is especially true so far as the control of digitalization in auricular fibrillation is concerned.

Where there is a very rapid auricular fibrillation, the pulse deficit may be very marked, and the physician may assume that because of a slow radial pulse rate, the patient is responding to his digitalis therapy. In fact, vomiting and other signs of digitalis overdosage may cause him to stop

his medication, feeling that digitalis saturation has occurred. A typical tracing of such a condition is presented below.

In a case such as this, the physician may be readily deceived as to the true state of affairs, in that *more*, rather than *less*, digitalis should be administered. Much valuable information will be lost in the study of auricular fibrillation, if only electrocardiographic studies are made. The return of compensation under digitalis or other therapy can be

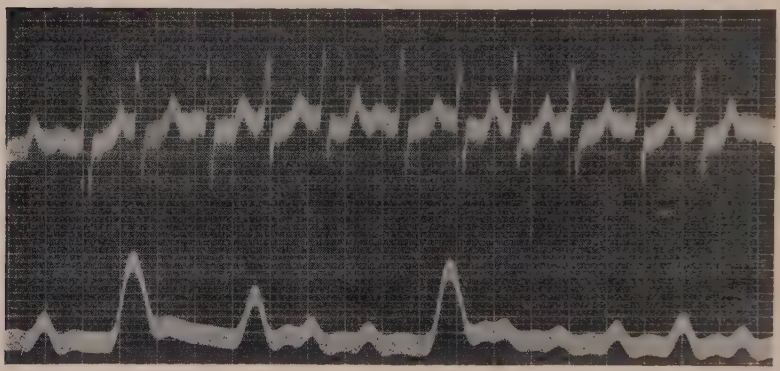

Fig. 66.—Simultaneous polygraphic and electrocardiographic tracings of rapid auricular fibrillation. Note that the ventricular rate is so rapid and irregular that incomplete filling occurs and several contractions of the left ventricle are made before a single impulse is given to the peripheral circulation. The radial pulse in such cases is no index of cardiac activity.

easily indicated by the disappearance of the pulse deficit; without radial pulse tracings such pulse deficits can not be determined.

MIXED FLUTTER AND FIBRILLATION

In the pathological development from normal sinus rhythm to auricular flutter, and thence to auricular fibrillation, many intermediate conditions may be found. While these conditions are usually considered theoretically as separate entities, in actual clinical experience, these changes may be, and frequently are, found in the same patient at different times. Complete auricular flutter or fibrillation

are not ushered in spontaneously; there are usually pre-existing phenomena which take place before the actual onset of the disturbance. Paroxysmal attacks of auricular flutter, lasting for a few seconds or minutes, usually occur before the complete development of continuous flutter. Such records may show a normal sinus rhythm interrupted by a short attack of auricular flutter.

In a similar manner, complete auricular flutter may show attacks of fibrillation. Alternation of flutter and fibrillation

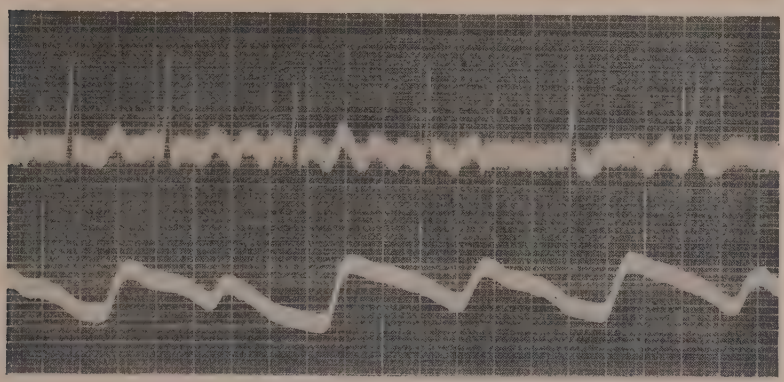

FIG. 67.—Impure auricular flutter and fibrillation. Woman, age 28, rheumatic mitral stenosis. Note intervals of auricular flutter and coarse auricular fibrillation. Two weeks later, under digitalis therapy this patient showed a coarse auricular fibrillation.

is not an uncommon electrocardiographic finding, and the transition from one into the other is frequently seen in mitral stenosis. Such alternation of rhythm is known as *impure flutter and fibrillation.*

Finally, auricular fibrillation may show changes from fine to coarse circus movement.

ETIOLOGY OF AURICULAR FIBRILLATION

From the point of view of pathologic physiology, auricular flutter and fibrillation can be regarded as the end results of several specific processes. In a study of 200 cases

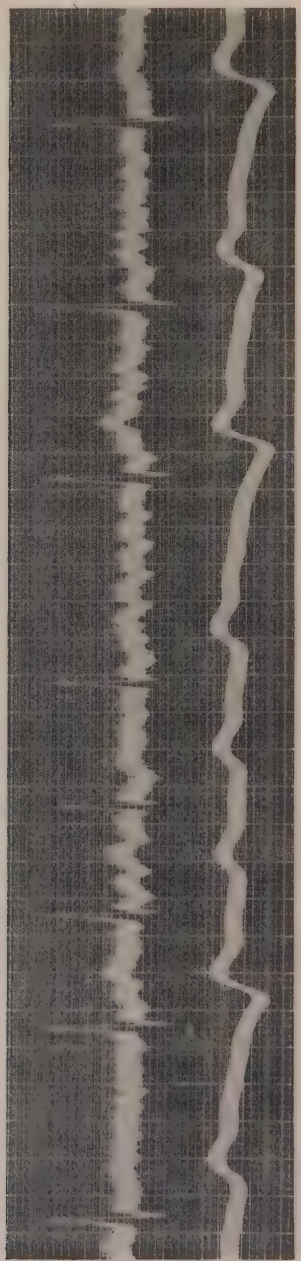

FIG. 68.—Alternating coarse and fine circus movement in auricular fibrillation.

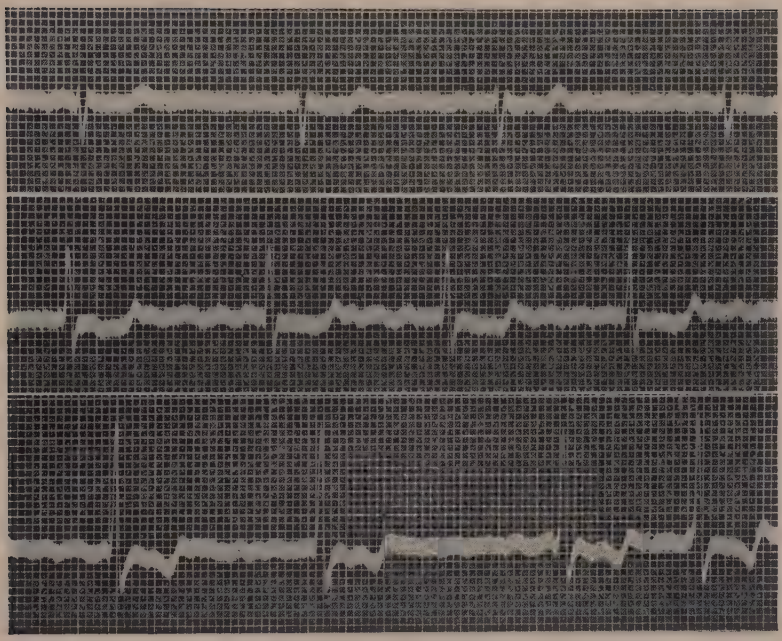

FIG. 69 A.—Auricular fibrillation in a case of rheumatic mitral disease. Woman, age 31.

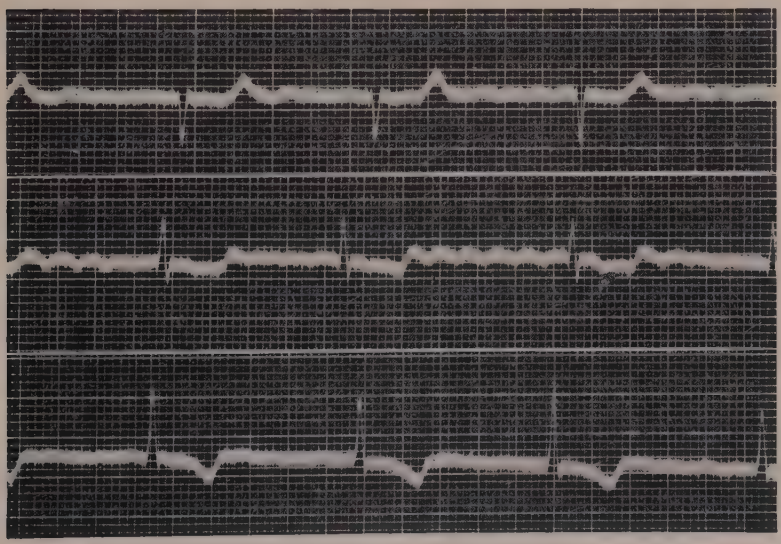

FIG. 69 B.—Auricular fibrillation in extreme mitral stenosis.

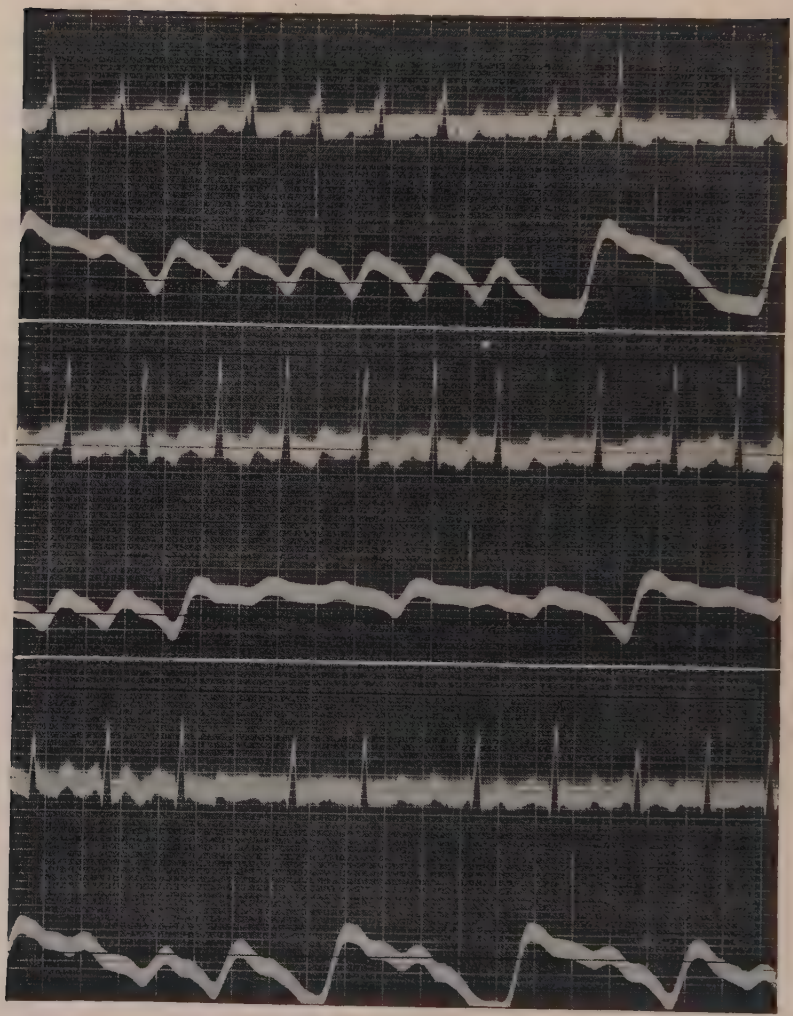

FIG. 70.—Auricular fibrillation in a case of hyperthyroidism. Woman, age 38.

of auricular fibrillation, Parkinson showed that five etiologic groups could be determined; the first, and by far the greatest number, were due to rheumatic heart disease, and especially those with valvular defects like mitral stenosis. Secondly, those cases arising from hyperthyroidism; third,

those occurring as a result of coronary artery disease; fourth, those associated with hypertension; and fifth, an ill-defined group, said to be due to myosclerosis with hypotension and with renal involvement.

In a general way, the alert electrocardiographer may at

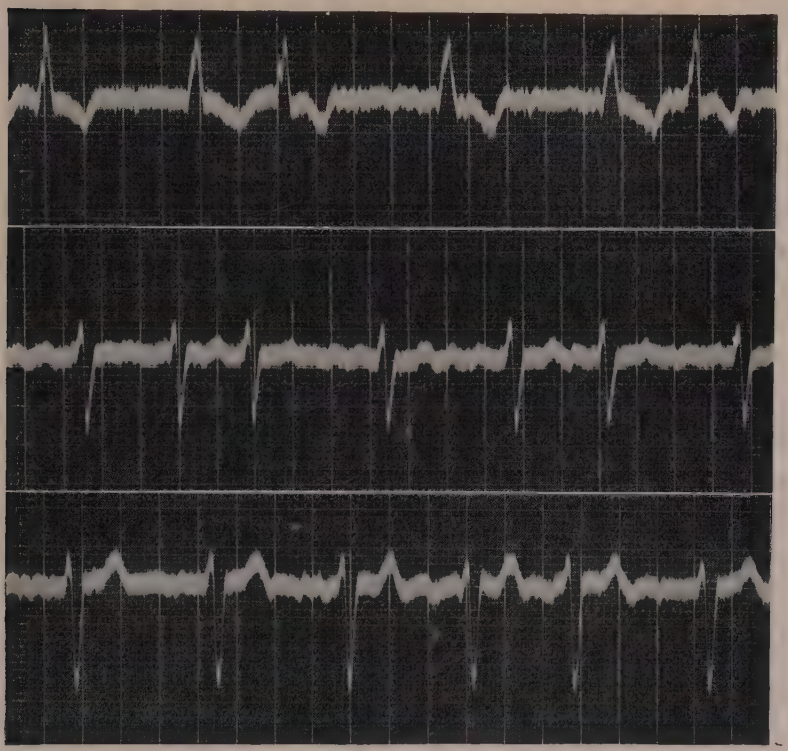

FIG. 71.—Auricular fibrillation in a case of coronary artery sclerosis. Man, age 62.

times be able to deduce the specific origin in a case of auricular fibrillation. Typical illustrations of each of the five types of auricular fibrillation are given below.

Rheumatic mitral disease. This tracing shows an auricular fibrillation with a right axial deviation of the heart; with the exception of certain rare diseases affecting the right

side of the heart, such right axial deviation is seen most often in rheumatic mitral stenosis. Rheumatic heart disease usually shows rapid auricular fibrillation; the discovery of these two conditions should suggest the etiology of the fibrillation.

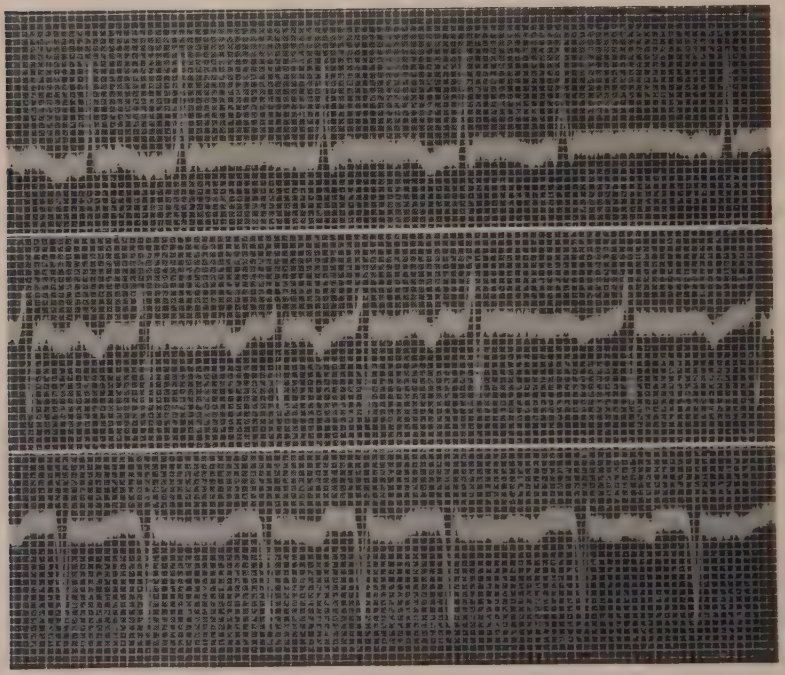

FIG. 72.—Auricular fibrillation in a case of hypertension. Man, age 78.

Hyperthyroid disease. It will be noted in this tracing that the ventricular rate is very rapid, that there is a very coarse circus movement, and that there is a tendency to a right axial deviation.

Coronary artery disease. This case shows a rather slow ventricular rate, a somewhat widened QRS complex, characteristic inversion of the T waves in the significant leads, and a moderate left axial deviation of the heart. There is also a very fine circus movement.

Hypertension. This case shows a slow ventricular rate, a marked left axial deviation of the heart, and a fine circus movement. Occasionally inversion of the T waves may be noted in one of the significant leads.

Myosclerosis. This tracing shows a moderately rapid ventricular rate, with delay and arborization of the QRS com-

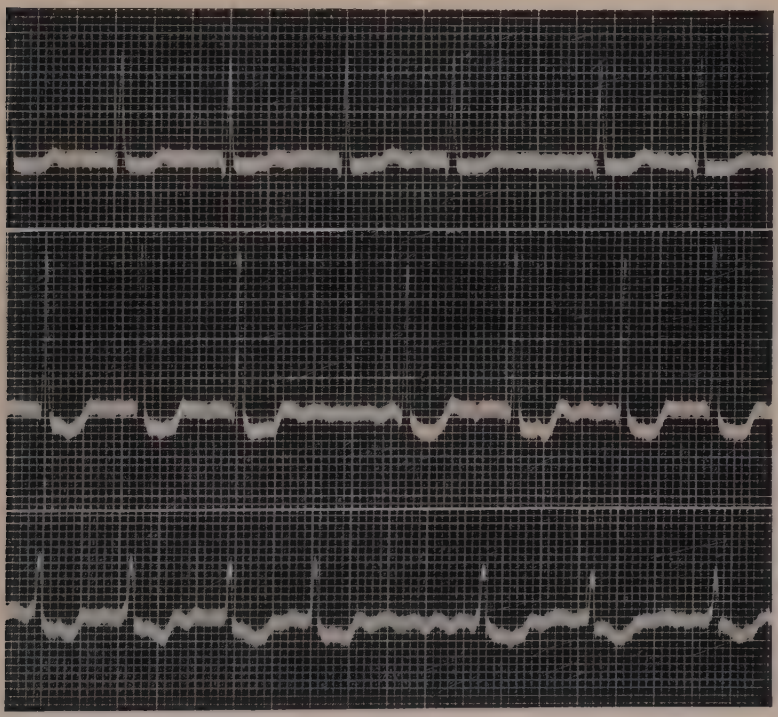

FIG. 73.—Auricular fibrillation in a case of myosclerosis. Man, age 62.

plex, no axial deviation of the heart, and a rather coarse circus movement.

In typical cases, like those above, the deductions made from the tracings may be of considerable importance from a therapeutic standpoint; it must not be inferred, however, that all cases of auricular fibrillation are thus readily ana-

lyzed, for two conditions may be present at the same time. This is especially true in regard to hypertension and coronary artery disease and myosclerosis. Occasionally, in rheumatic heart disease where there is also involvement of the aortic valves, there may be a left instead of a right axial deviation of the heart.

ELECTROCARDIOGRAPHIC CONTROL OF DIGITALIZATION IN AURICULAR FIBRILLATION

Few drugs exhibit the specific response in therapy that digitalis does in decompensated rheumatic auricular fibrillation. As a matter of fact, the use of digitalis in this condition represents one of the oldest cardiac remedies. Notwithstanding this, each year finds some new knowledge added to the efficacious administration of this valuable substance. In this regard, the electrocardiographic study of such patients receiving digitalis, constitutes a most accurate control in their management. This is due to the fact that digitalis causes certain electrodynamic changes that are easily discovered.

It has been demonstrated by Pardee, Eggleston, and others, that active digitalization is accompanied by typical alterations in the T waves, especially in Lead I, but also in leads II and III. This T wave alteration takes the form of the so-called bowed type. (See tracing No. 107 on page 165.)

The discovery of the T wave change should be used as a guide for diminution in the digitalis dosage. If digitalization proceeds beyond this point, the typical signs of overdosage will appear. Clinically, these are seen as severe occipital headaches, vertigo, nausea, and vomiting. Sometimes disturbances of vision are complained of; this is especially so in regard to color perception, the patients complaining of a greenish hue being cast on all bright objects. The radial pulse becomes slow, and a bigeminal rhythm is noted.

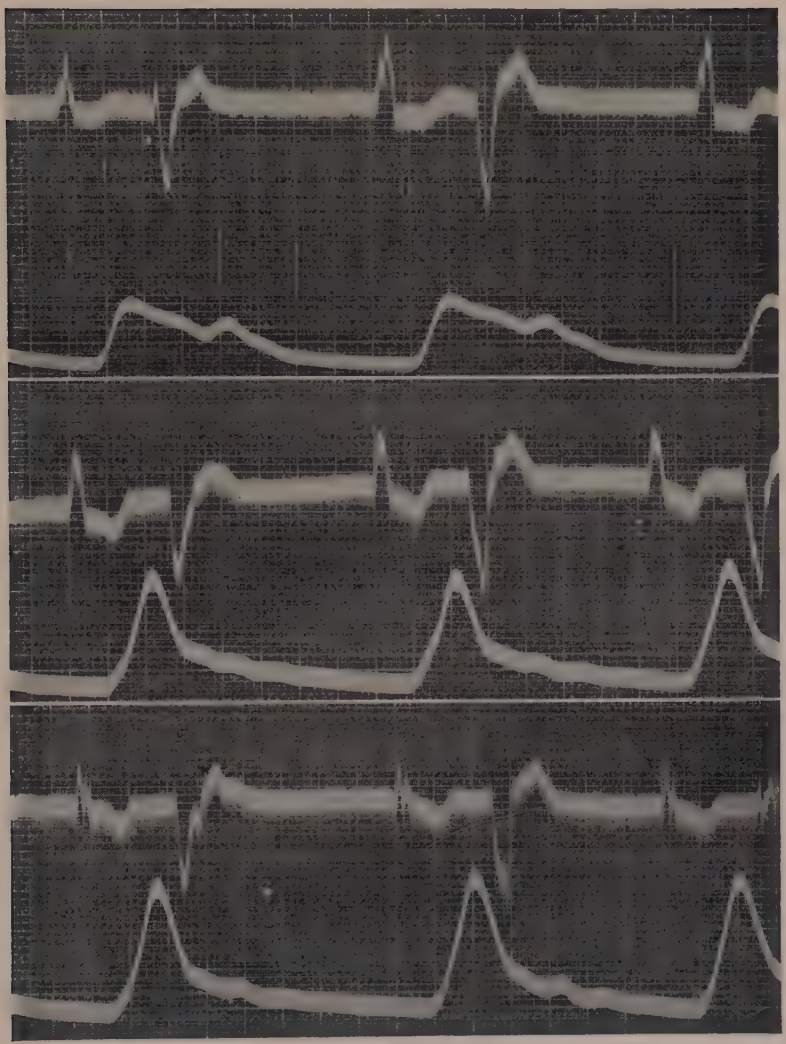

FIG. 74.—Bigeminal rhythm. Woman, age 28. Decompensated rheumatic mitral auricular fibrillation. Tenth day after intensive digitalis therapy. Note that a left ventricular extrasystole follows each normal beat. The T waves show characteristic digitalis inversion.

FIG. 75.—Digitalis overdosage in auricular fibrillation with alternating ventricular extrasystolic arrhythmia. Note here that the normal beat is followed first by a left ventricular extrasystole and then by a right ventricular extrasystole; occasionally the order may be reversed or there may be several alternations.

FIG. 76.—Digitalis overdosage in auricular fibrillation. Many extrasystoles occurring in a series first in one ventricle and then in the other. This condition represents an extremely dangerous point in digitalis therapy; when a long series of such extrasystoles occur very rapidly, complete mechanical breakdown of the ventricles may occur with cessation of the peripheral circulation and syncope, prolonged unconsciousness and instantaneous death.

Electrocardiographically, this bigeminal rhythm is found to be a normal ventricular complex followed by a ventricular extrasystole.

If digitalization is pushed beyond this point, toxic dissociation of the heart may result. Under such conditions there may develop an extrasystolic arrhythmia involving first one ventricle and then the other. If this extrasystolic arrhythmia persists longer than a few seconds complete ventricular asystole may occur with instantaneous death.

Excessive overdosage of digitalis may give rise to frequent extrasystoles. If a long series of such extrasystoles develop, ventricular fibrillation and subsequent death may ensue.

While it is true that overdosage to such a degree, rarely occurs under adequate medical supervision, it is also equally true that under the modern routine of massive digitalization the step from saturation to overdosage can readily occur. If frequent electrocardiographic studies are made for the purpose of controlling digitalization, the discovery of the characteristic T wave alterations may prevent the patient from undergoing the disagreeable consequences attending overdosage with its nausea, vomiting, and headache, and not the least—the gastrointestinal reactions which may persist long into convalescence.

In brief, electrocardiographic control of active digitalization may avoid many of the disagreeable, and often dangerous sequelae of overdosage; not only in the acute phases, but also in the avoidance of the disastrous gastrointestinal upsets, which once established in such cases, are hard to overcome.

CHAPTER VI

DISEASES OF THE CONDUCTING SYSTEM

DISEASES OF THE CONDUCTING SYSTEM

The disturbances of conduction form a well defined group of cardiac disorders which are readily understood by reverting again to our simile of the internal combustion engine. The perfect functioning of such an engine demands that an electric impulse be carried without interruption and regularly from the generator to the spark plugs of the respective cylinders by way of the proper distributor. If an impulse fails to reach a cylinder at exactly the proper time and the proper intensity, the gasoline is not exploded and that cylinder fails to perform its special part in the chain of events.

In a very similar manner the conduction system and its possible disturbances may cause disfunction in the orderly contraction of the various chambers of the heart. As has been pointed out before, the conduction system of the heart is not a completed or continuous circuit. There is no special pathway from the sinus nodal pacemaker to the auriculoventricular node. The impulse is released from the pacemaker and spreads through the auricular muscle and reaches the lower node by way of a muscular lane or circuit. From the lower node to the final filaments of the Purkinje system, the impulse finds its way over a highly specialized system of conducting tissue. If we again take our analogy to an electric circuit, the pathway through the auricles may be compared to passing a current through a high resistance coil where there is subsequent delay in the rate of speed of the impulse, whereas a passage of the impulse through the bundle tissues of the heart may be compared to an electric current being sent over a non-resistant copper cable.

In point of fact, it has been experimentally determined that the impulse requires more than four times as long to pass through myocardial tissues as through the specific conduction system. With these thoughts in mind, it may be

readily understood that disturbances in conduction from the pacemaker to the auriculo-ventricular node may occur in any condition in which the auricular muscle is changed from normal. These changes may occur in normal auricles; they may also occur temporarily in acute pathologic processes, and finally, they may be of a permanent or chronic character when unrecoverable processes have taken place in the auricular musculature.

In all these conditions, regardless of the etiology, the muscular tissue does not carry the electrical impulse as rapidly or as regularly or, perhaps not at all, as compared to normal muscular tissue. Clinically, and from an electrocardiographic point of view, these various changes have been classified under different degrees of *heart block*. Where the impulse is merely delayed in its passage through the auricle, the condition is known as *first degree heart block*. When the impulses are blocked to such an extent that the impulse from time to time fails to reach the lower node and thus initiate a ventricular response, the condition is known as *second degree* or *partial heart block*. Finally, in those conditions in which the impulse never reaches the lower node and where the lower node has now initiated its own impulse for ventricular contraction and thus becomes independent of auricular contraction, the condition is known as *third degree heart block* or *complete dissociation*.

FIRST DEGREE HEART BLOCK

First degree heart block is readily indicated by a lengthening of the P-R interval above its upper normal limit of 0.20 seconds. Formerly thought to be a rather unusual condition it has been shown within recent years to be frequently associated with most of the acute infectious diseases, and is especially common in acute rheumatic fever, diphtheria, scarlet fever, pneumonia, and in certain types of influenza. It may be occasionally noted in certain very toxic acute con-

ditions like tonsillitis and erysipelas, and whenever found is unmistakable evidence of myocardial involvement. *Its importance to the clinician lies in the fact that it is a definite indication for prolonged convalescence, for it is believed that*

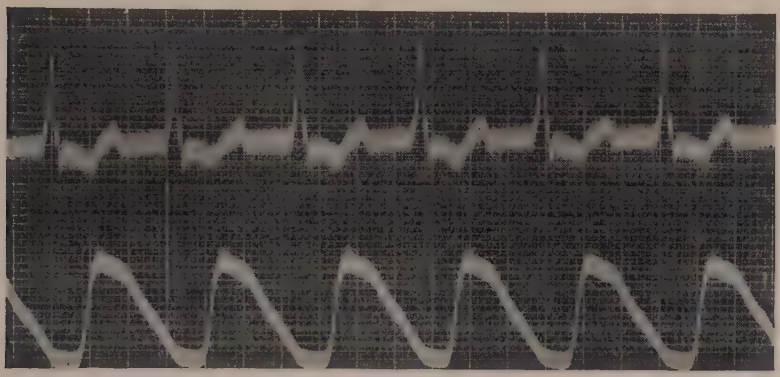

FIG. 77.—Prolongation of P-R interval. (First degree heart block.) 0.32 seconds in a case of pneumonia.

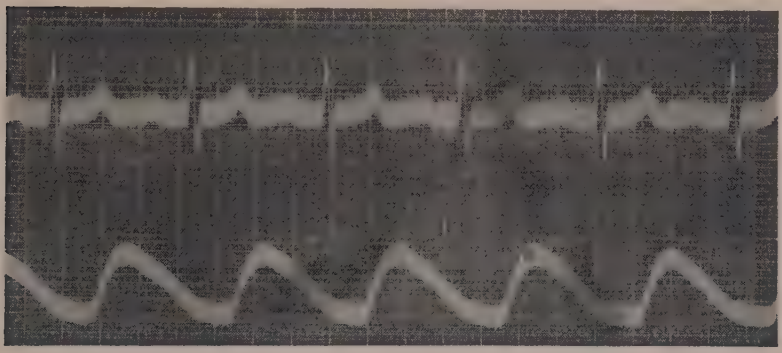

FIG. 78.—Same case of pneumonia taken 14 days later. The P-R interval now measures 0.22 seconds.

heart strain at this time may cause irremediable myocardial damage.

Delay in the P-R interval to 0.28 and 0.32, are rather common in such acute infectious diseases; this condition may very quickly disappear.

Where, however, the P-R interval is raised to 0.40, the

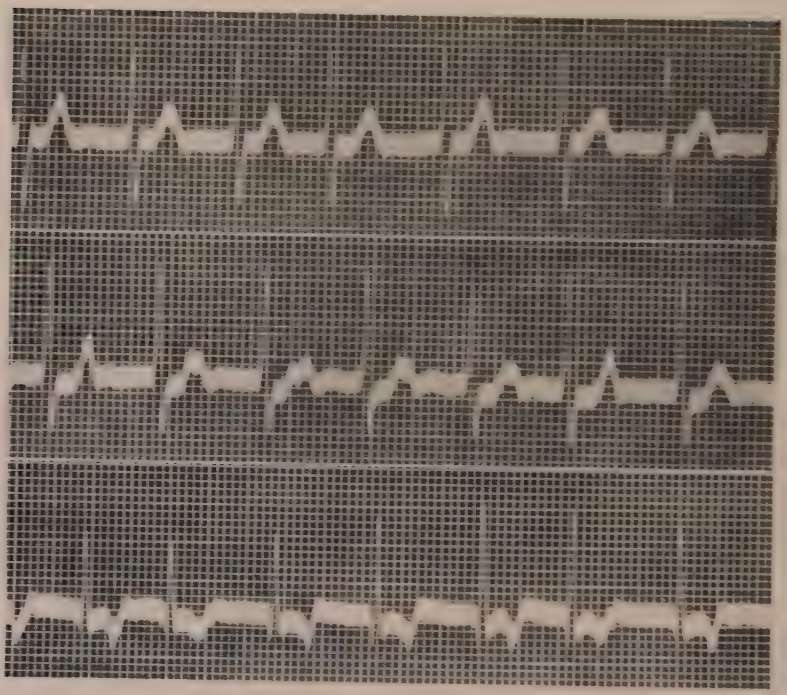

FIG. 79.—Records taken from a boy, age 10, during an acute attack of rheumatic fever, showing a prolongation of the P-R interval to about 0.40 seconds. The P-R interval is so long that it coalesces with the T wave of the preceding beat. This record is interesting in that it may be mistaken for auricular fibrillation, as the QRS complexes are apparently not preceded by a P wave. Examination of the T waves, however, show them to be constantly changing in size and configuration, and from time to time the P wave can be seen emerging from the T wave.

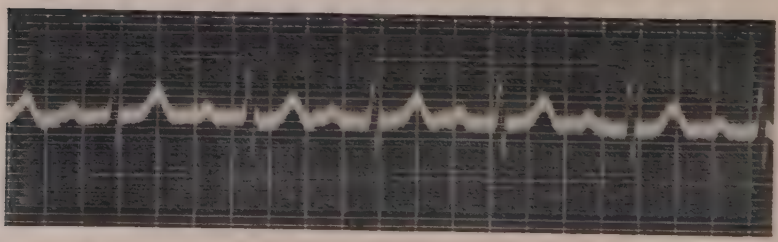

FIG. 80.—Same case two months later. The patient was discharged as clinically recovered six weeks previously. P-R interval still prolonged to 0.32 seconds.

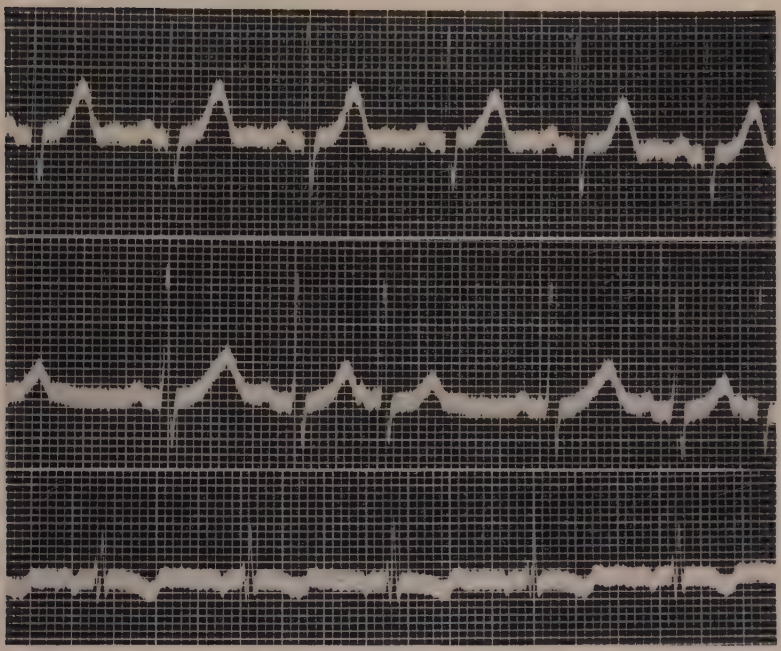

FIG. 81.—Same case one year later. The P-R interval now measures 0.18 seconds. Note the presence of auricular extrasystoles.

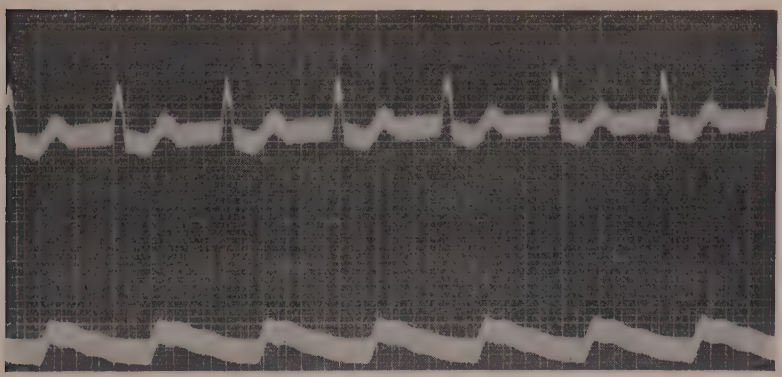

FIG. 82.—Prolongation of P-R interval to 0.42 seconds in patient with myosclerosis.

condition is not so readily recovered from and permanent damage may have been sustained by the auricular tissue.

Permanent lengthening of the P-R interval is also seen in those hearts exhibiting chronic degenerative changes with vascular and myocardial sclerosis.

Graphically the condition is very easily recognized and no special note need be added in this regard.

SECOND DEGREE OR PARTIAL HEART BLOCK

When the passage of the impulse through the auricular musculature is delayed to such a point that the next impulse is released from the sinus node before the first one reaches the auriculo-ventricular node, the second impulse may find the junctional tissue in such a refractory stage that the impulse calls forth no ventricular response. In this way there is a complete loss of a ventricular contraction, and it can be noted at the radial pulse by an intermission in the rate.

Electrocardiographically the condition is easily recognized.

Occasionally there are other complex factors in addition to the mere delay of the impulse through the auricular tissue. These other factors involve a certain fatigability of the auricular muscle in such a way that after an impulse had passed through it, it permits the next impulse to pass through at a lessened rate, and a third at a still lesser rate. This phenomenon, known as "The Wenckebach Period Syndrome," finally reaches a point in which the fatigability of the auricle is so great that no impulse can be passed through it at all. At such time there is no ventricular complex. It can be mathematically established that the rate of fatigability proceeds at a rate which is constant; in this way there will be a regular dropping out of a ventricular complex. The regular omission of such complexes is spoken of in relation to the auricular beats. Thus,

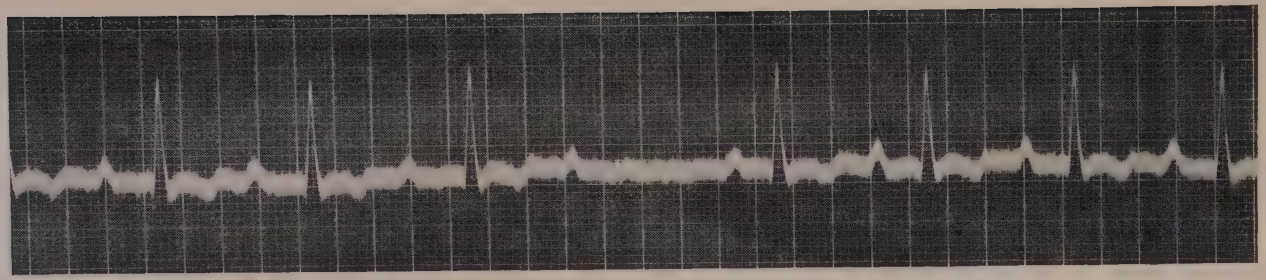

FIG. 83.—Second degree or partial heart block. In this case a single ventricular complex has been dropped. Note that the P waves are occurring regularly. At beat 4 the auricular complex is not followed by a ventricular complex. This condition is sometimes known as "dropped beat," but should not be confused with the clinical expression of "skipped beat." This latter phrase is graphically inaccurate in that there is no skipping of the beat at the radial pulse, but as a matter of fact, there is an extra or premature beat followed by a compensatory pause. It is this compensatory pause which gives the impression of the so-called skipping of the beat.

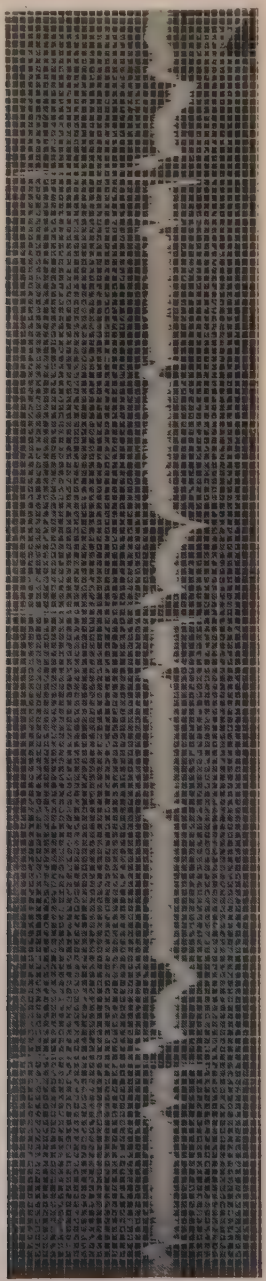

FIG. 84.—Three to one heart block. Note three P waves to every QRS complex.

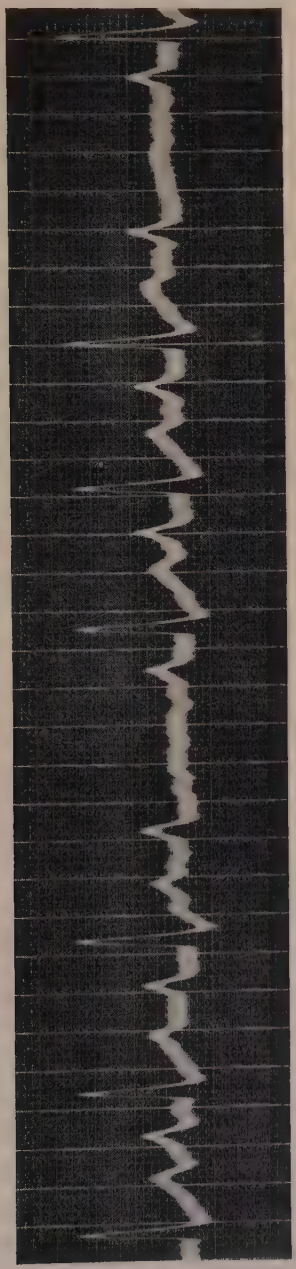

FIG. 85.—Four to three heart block.

for example, if every fourth ventricular beat is lost, a four to three ratio is established. In other words, the auricle has beaten four times and the ventricle three. Certain ratios are more common than others; in our experience three to two, four to three, and five to four are the most frequent. Under extreme conditions as described above, the fatigability of the auricle may be so great that only every other impulse passes through; here we get the so-called, *two to one rhythm.*

While the Wenckebach phenomenon explains the omission of a ventricular complex at a given regularity, all of the factors demanded by the theory may not be found in the tracing. Theoretically, the condition demands that the P-R interval grow progressively longer until a ventricular complex is dropped out, and the next P-R interval is within normal limits. This is said to be due to the fact that the auricle has recovered from its fatigability during the dropped ventricular beat.

Many times, however, second degree or partial heart block with a very regular four to three or five to four ratio can occur without evidence of the progressively increasing P-R interval. The phenomenon is explained in these conditions as being due more to fatigability on the part of the junctional tissue than to the auricular musculature. Cases are not infrequently seen in which the P-R interval may be within normal limits and yet a partial block has developed; the disturbances under these conditions must lie in the junctional tissue.

THIRD DEGREE OR COMPLETE HEART BLOCK

The familiar physiological laboratory experiment in which the auricles and ventricles of a frog are dissociated is well known to every medical student. Destruction of the junctional tissue by any pathologic process results in a condition in which impulses from the pacemaker are blocked

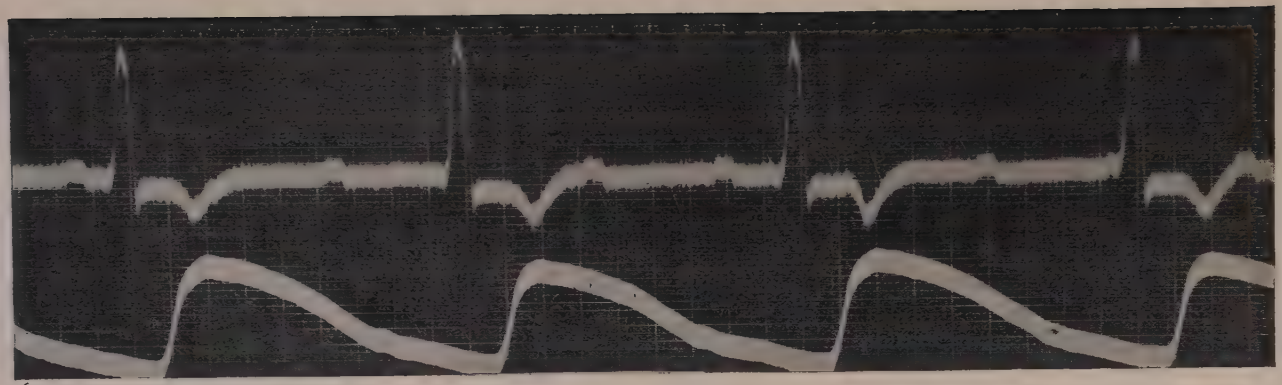

FIG. 86.—Third degree heart block or complete auricular and ventricular dissociation. Note here that the P waves are occurring regularly at a rate of 78 beats per minute, while the ventricular rate is more or less regular at 34 beats per minute. Electrocardiographically the condition is readily recognized by what appears to be a constantly changing P-R interval. There is, however, no P-R interval because the impulses from the auricle are blocked at the atrio-ventricular junction and never reach the ventricle. This condition must be differentiated from alternation of the pacemaker (see Fig. 42). In the latter case impulses do pass through from the auricle to the ventricle and the auricular rate is very slow.

before they reach the auriculo-ventricular node. Because of the peculiar life saving mechanism already described in a previous chapter, the lower node now assumes the rôle of pacemaker for the ventricles. An interesting condition now arises in which both pacemakers of the heart are working simultaneously, each governing its respective portion of the heart without relation to each other. The auricular rate, as governed by the sinus node, is usually at its normal frequency of 72 times per minute, while the ventricles are contracting at the lower nodal rate of about thirty-two beats per minute. This condition is known as *complete dissociation* of auricular and ventricular rhythm, and is a pathologic condition of the heart fruitful of much academic speculation.

In our consideration of complete dissociation we have assumed that the pathological condition which has caused the third degree heart block has localized itself exclusively to the junctional tissue of the heart in much the same manner as the original Gaskell experiment on the frog's heart. While it is true that the diseased condition may pick out a single portion of the heart, it is probably more often the case that the same disease process has also involved other portions of the heart. For example, the auricles may be affected by a spreading of the pathology in such a way that the musculature is affected. Points of irritability arise that readily become ectopic foci for new areas of contraction. Extrasystoles, flutter, fibrillation, or any mixture of these, may be concomitant conditions with heart block. The ventricular portion also may undergo associated changes in which one or the other of the main bundle branches may be affected, or finally there may be a *Purkinje fibre block*.

In addition to the concomitant conduction disturbances that may occur in complete heart block, there may be also the various conditions which affect the two pacemakers, so that a very complicated picture of pacemaker disease

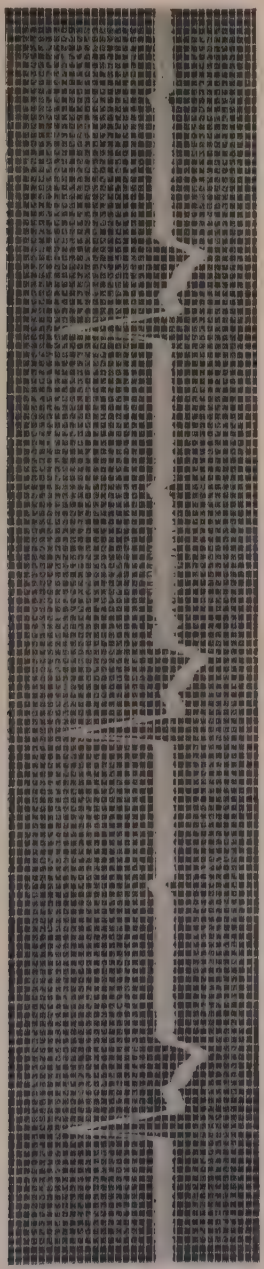

FIG. 87.—Complete dissociation in a case of coronary artery sclerosis.

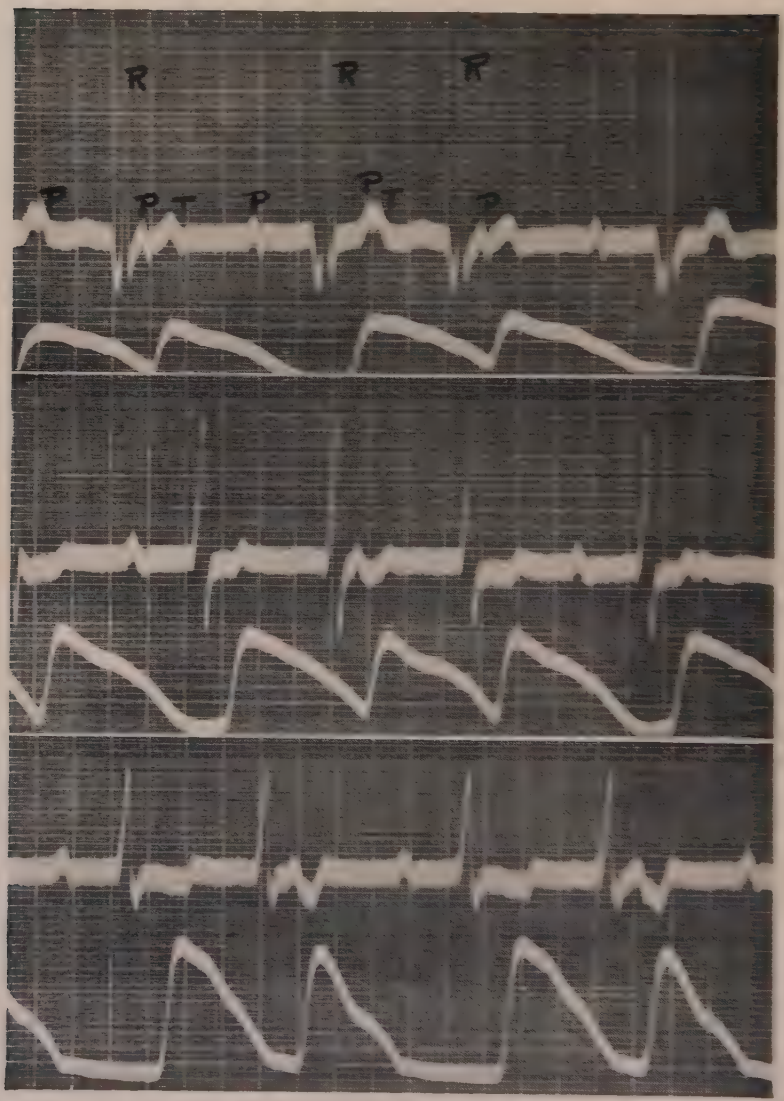

FIG. 88.—Complete dissociation with unusual contour of rhythm in Lead III

with its peculiar changes of impulse production, together with the dissociation of auricles and ventricles may occur. The picture presented under these conditions may prove to be almost impossible of deciphering.

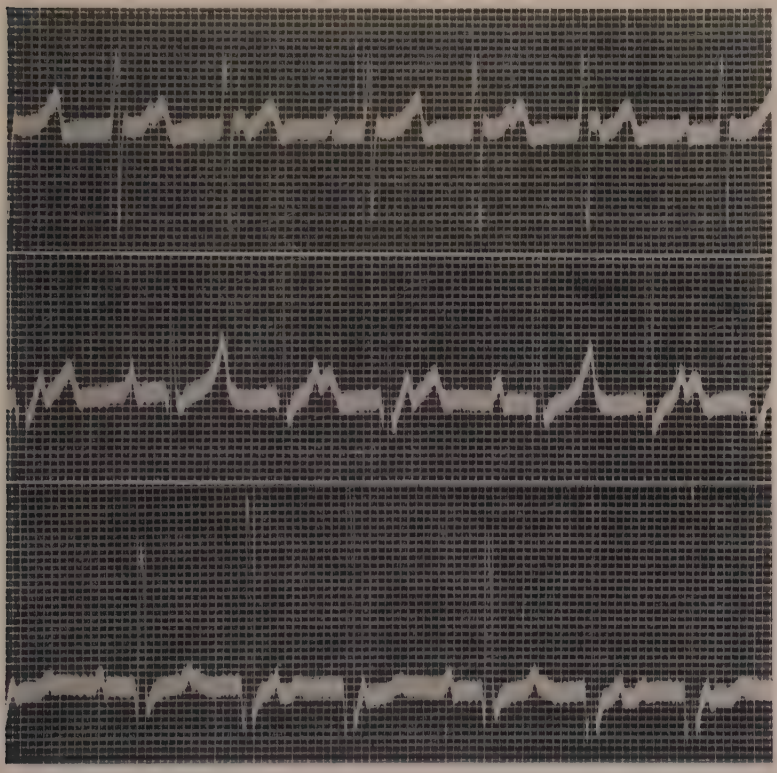

Fig. 89.—Alternating complete and incomplete heart block.

Probably no other condition will tax the keenness and patience of the electrocardiographer more than the tracings obtained in such cases, but to one interested in the problems of the cardiac mechanism, their occurrence should prove to be a constant source of delight in their unraveling.

BUNDLE BRANCH BLOCK

Anatomically it has been demonstrated that the Bundle of His divides into two main branches of about equal size; the one piercing the interventricular septum and controlling the right ventricle, while the other controls the left. Pathologic conditions causing destruction of one of these main branches are readily discernable by cardiographic study. If, for example, a disease process has affected the continuity of the right main bundle branch, few or no impulses will be able to reach the right ventricular musculature. The impulse for contraction passing rapidly down the main bundle simultaneously spreads over both branches; where one branch is damaged, the impulse passes down the other in its usual fashion and when it has reached the terminal filaments of the Purkinje system, the impulse is carried over by way of the muscle into the right side of the heart.

As we have shown before, the rate of propagation of the stimulus is more than four times as fast through the specialized conducting system as through the heart muscle. The spread of the wave, therefore, is rapidly accomplished through the left ventricle but must be considerably delayed through the right. From our previous knowledge of the electrodynamic phenomena of the heart, we can theoretically anticipate how the ventricular complex will be altered in this condition. Inasmuch as the impulse must be spread by way of the left ventricle the complex must assume a left ventricular predominance in Leads I and III, and inasmuch as the spread of the impulse must be by way of musculature, the transmission of the impulse must be delayed. This will be indicated electrocardiographically by a delay in the QRS complex. Hence, the discovery of a markedly delayed QRS complex with a left axial rotation of the heart should indicate that there has been a disturbance or a destruction of the conducting system to the

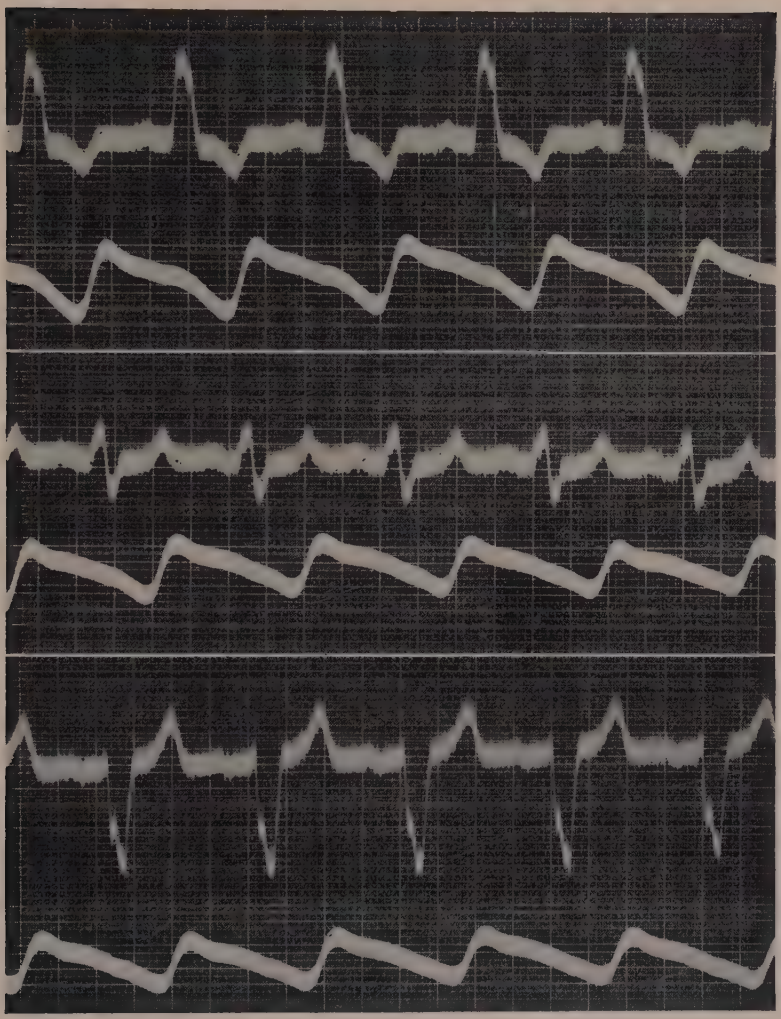

FIG. 90.—Right main bundle branch block. Note here that the ventricular complexes are delayed in transmission time to 0.14 and 0.16 seconds.

right ventricle. Such, as a matter of fact, is the case, and experimental work has shown that cutting the right bundle will give the above described configuration.

Exactly similar processes occur when the damage has de-

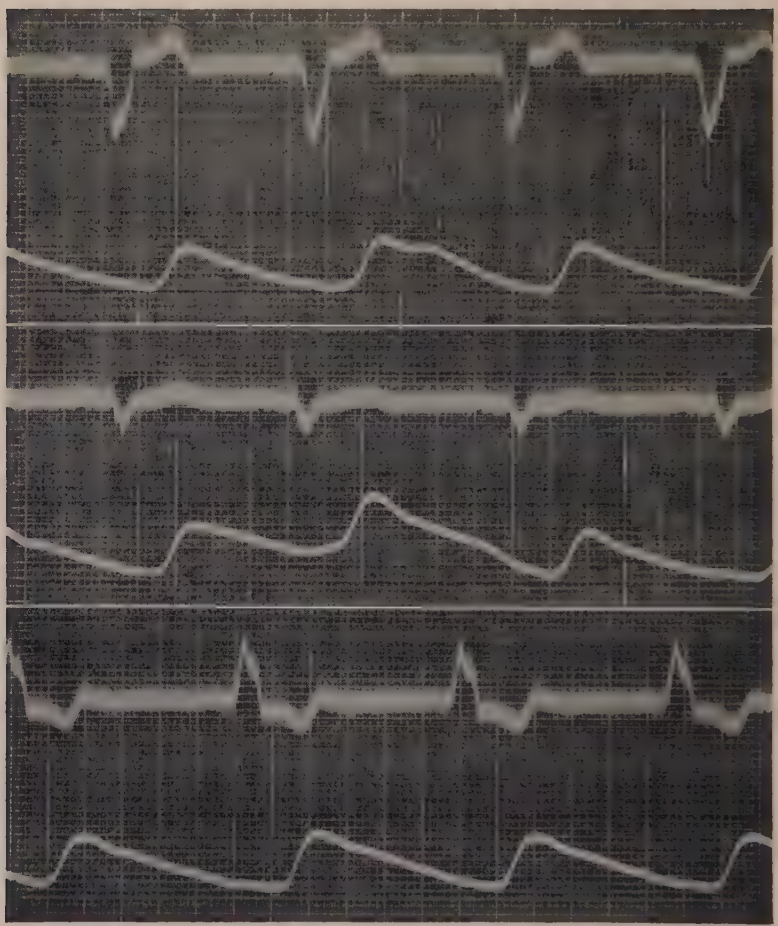

FIG. 91.—Left main bundle branch block.

veloped in the left bundle. The excitation wave must now pass first through the right ventricle before it can pass by a muscular pathway to the left ventricle. The QRS complex now assumes a right ventricular predominance with a similar delay in conduction.

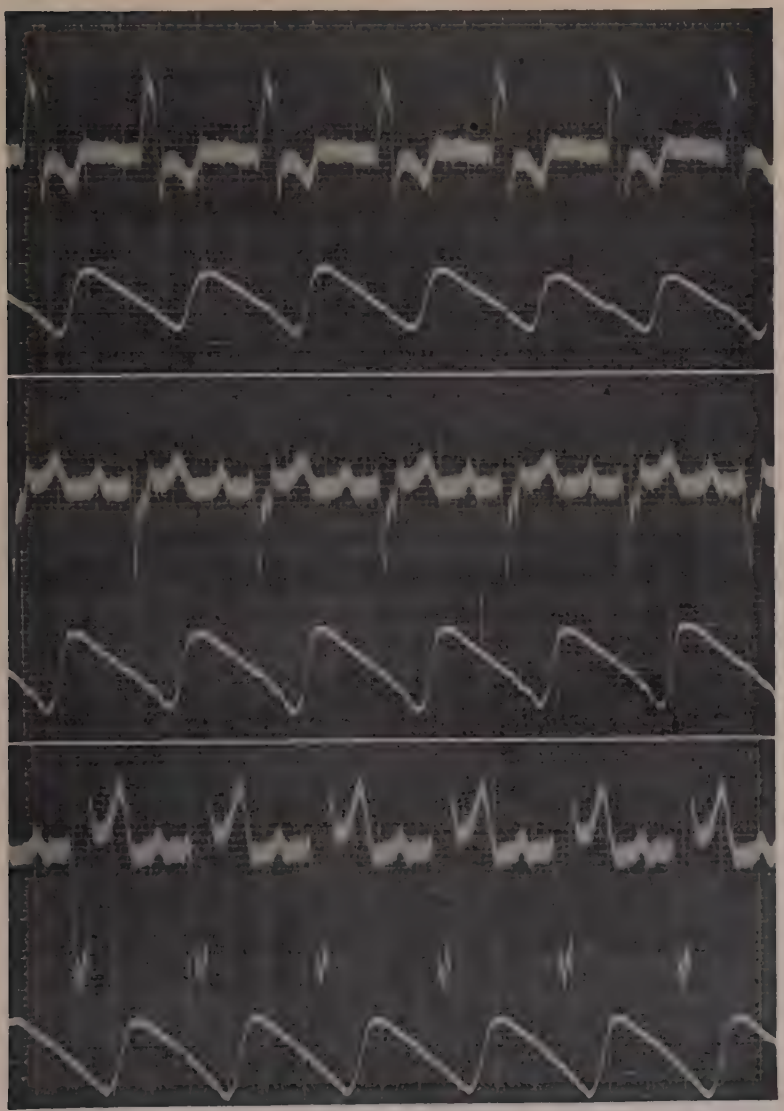

FIG. 92.—Arborization block. Note the fine feathering or notching of the QRS complexes.

TERMINAL FIBRE BLOCK, ARBORIZATION BLOCK PURKINJE SYSTEM BLOCK

Very frequently degenerative changes occurring in the musculature of the heart involve small areas of the Purkinje fibre network. If such a destruction takes place in an active portion of the ventricles, especially at the region of the apex, there may be an irregular rate of propagation of the impulse to the neuro-muscular junction. This will be indicated electrocardiographically by a very fine notching or feathering of the QRS complex on its upstroke and also downstroke.

CHAPTER VII

DISEASES OF THE VENTRICLES

DISEASES OF THE VENTRICLES

Ventricular Extrasystoles

Ectopic foci developing in the ventricular walls, release a stimulus for contraction which are forced to follow an unusual pathway through the various elements making up the contracting chambers of the heart. This unusual pathway presents a greater electrical resistance than that met with by the normal stimulus traveling over the specialized conducting system. For this reason, there is a very definite delay in the impulse as it passes through the ventricles.

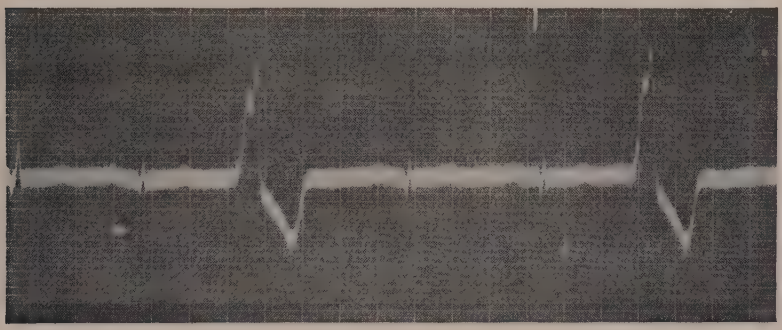

Fig. 93.—Right ventricular extrasystoles.

This delay may be two or three times as great as that required by the normal QRS complex. Moreover, the spreading of the complex may be accompanied by notching or splitting of the waves, suggesting an unusual pathway. Certain bizarre configurations may be given to the complex, distorting it to such an extent, that it is recognized with difficulty as being similar to the normal QRS complex. The terminal deflection of the ventricular complex, seen in ventricular extrasystoles, is usually opposite to that taken by the initial deflection. The terminal deflection resembles more closely the T wave of the normal ven-

tricular complex than the initial deflection resembles the QRS complex.

When the ectopic focus originates in the left ventricle, especially at its base, the initial deflection assumes a configuration of the levogram type, inasmuch as the impulse makes its first excursion through the left ventricle before the spread of the excitation wave reaches the right ventricle. The QRS complex is therefore, projected downward and the T wave is upward. (See Fig. 51.)

When, however, the ectopic focus arises at the base of the right ventricle, the impulse in following a pathway through the right side first, imparts a dextrogram phase to the configuration. The initial deflection in this case will be upright, and the T wave will become inverted. (See Fig. 45.)

There is considerable experimental evidence to show that the spread of the excitation wave through unaccustomed pathways can be demonstrated by the use of certain well defined mathematical formulæ. These formulæ explain why the ventricular complex assumes different configurations, depending upon the site of the ectopic focus. Such explanations do not lie within the scope of this presentation, but it should be noted that different values must be given to each of the three leads. The most marked differences for these values appear in Leads I and III and the least between II and III. For this reason, the configuration of the initial ventricular complex may assume a very different form in Lead I, compared to Leads II and III. Likewise, the terminal ventricular complex as seen in Lead I, may not be opposite to the initial deflection; both deflections may be upward and downward together.

According to the older classification, those extrasystoles which were different in the first lead from those found in the second and third, were known as *discordant* types of ventricular extrasystoles, while those which were similar to the configurations found in Leads II and III, were called *concordant* ventricular extrasystoles. Further study has

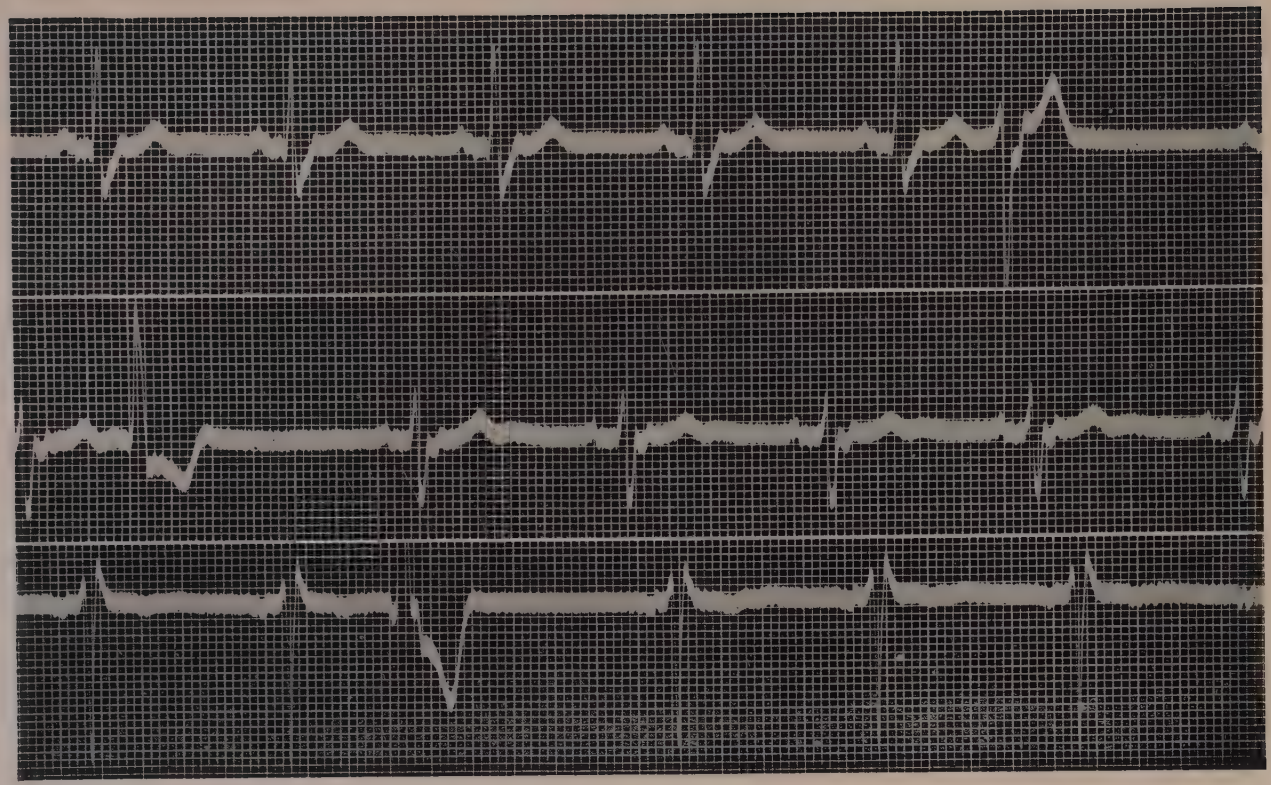

FIG. 94.—Discordant right ventricular extrasystole. Note that the extrasystole in the first lead assumes the mirror image of that found in the third. In such cases the extrasystole is studied in the second and third leads only.

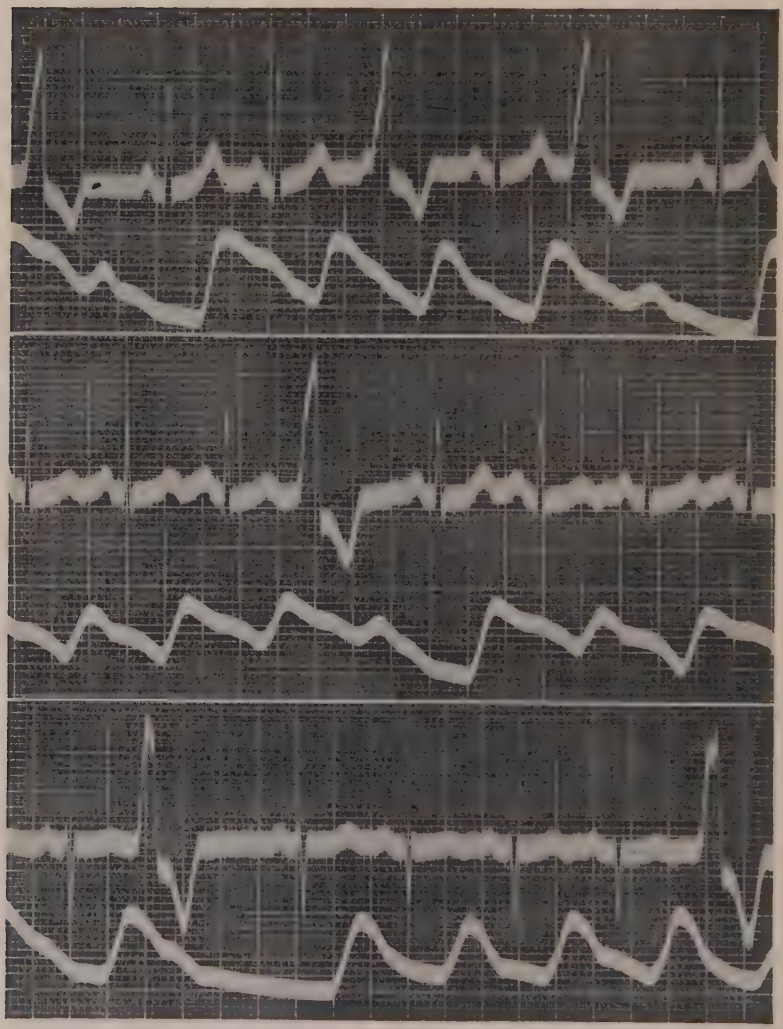

FIG. 95.—Right ventricular extrasystoles of the concordant type. Note that the configuration of the extrasystole is the same in all the three leads.

shown that a careful measurement of the initial deflection and its accompanying T wave may be used in theoretically demonstrating the point of origin of the extrasystoles.

In general clinical practice, however, the clinician need

concern himself only with the extrasystolic configuration as seen in the second and third leads. Ventricular extrasystoles are readily discovered in any tracing by their bizarre forms and their large excursions.

ALTERNATING LEFT AND RIGHT VENTRICULAR EXTRASYSTOLES FROM THE SAME FOCUS

Under certain ill-defined pathologic conditions, where an ectopic focus may be liberating impulses at a very rapid rate so as to produce a sequential extrasystolic rhythm, the phenomenon of alternation may occur. This takes place because of the ready fatigability of the unusual pathway over which the stimulus for the ectopic contraction must pass. When such a focus lies in tissues midway between the right and left ventricles, the impulse may pass first through one ventricle, and then through the other. In such a tracing, alternating right and left ventricular extrasystoles may be seen. (See Fig. 75.)

VENTRICULAR EXTRASYSTOLES FROM SHIFTING FOCI

Where the myocardium has undergone serious change, more than one ectopic focus may arise and become the source of stimulus production of extrasystoles. Under such conditions, extrasystoles may be found bearing different configurations, although assuming either a right or left sided origin. Such a finding may be regarded with especial significance, since it indicates widespread myocardial disease.

PULSUS BIGEMINUS OR COUPLED RHYTHM

Of especial interest is the occurrence of ventricular extrasystoles following a normal beat in a regular sequential rhythm. This condition is met with very frequently during

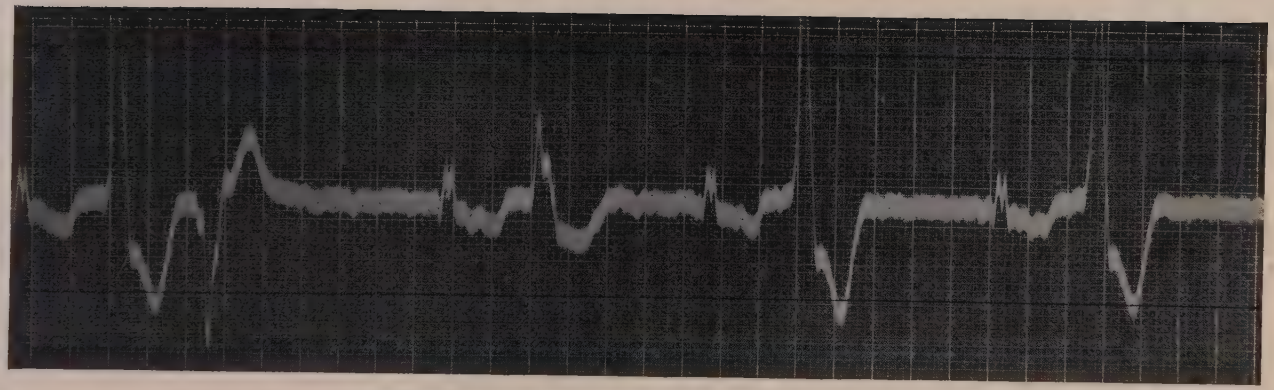

FIG. 96.—Ventricular extrasystoles from shifting foci.

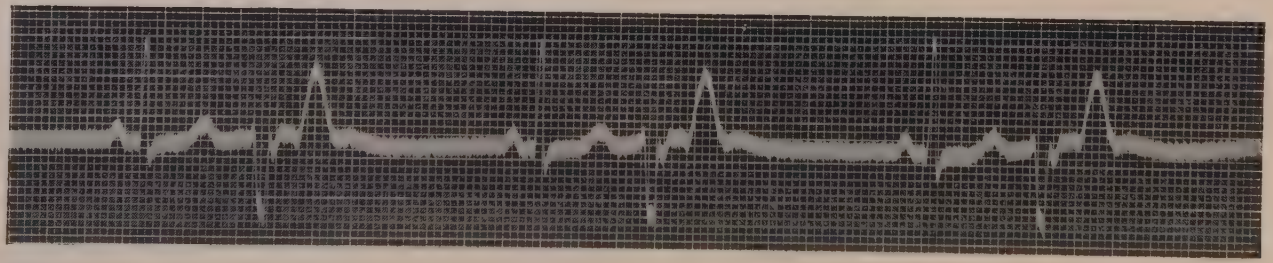

FIG. 97.—Pulsus bigeminus occurring during digitalization. Note that every normal beat is followed by a left ventricular extrasystole.

the process of digitalization, and is to be regarded as a definite sign of overdosage.

Where, however, such bigeminal rhythm is noted in patients not undergoing active digitalization, its finding can be regarded with especial significance, since it suggests extensive myocardial disease, and is found not infrequently in coronary artery disease with infarction.

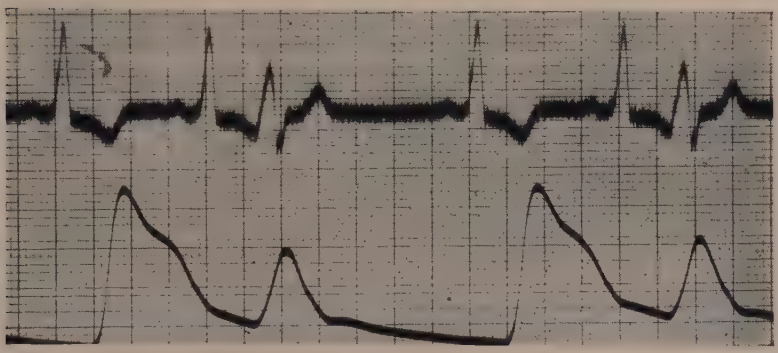

FIG. 98.—Coupled rhythm in coronary artery disease. Man, age 52, with typical anginal attacks for several months. This record was taken eleven days prior to sudden death.

Occasionally, a condition known as *pulsus trigeminus,* may arise, in which a ventricular extrasystole follows every second normal beat. The significance of this particular phenomenon is not well defined, but it may be regarded as very similar to that found in pulsus bigeminus.

VENTRICULAR PAROXYSMAL TACHYCARDIA

When one or several ectopic foci within the ventricles are capable of developing stimuli for successive ventricular contractions, a condition known as ventricular paroxysmal tachycardia may develop. Like all other paroxysmal tachycardias, the attack is ushered in abruptly and terminates with equal suddenness. The paroxysm may last for only the

duration of a few extrasystoles, or it may persist for several minutes or hours. The condition becomes especially serious when more than one ectopic focus develops, for at that time the danger of ventricular fibrillation is imminent. (See Fig. 57.)

VENTRICULAR FIBRILLATION

Fibrillation of the ventricles means complete suspension of the circulation and the onset of ventricular fibrillation is always accompanied by instant death. Few records have ever been obtained in this condition on human subjects, but experimental animal investigation has shown that ventricular fibrillation can be compared in many respects to the

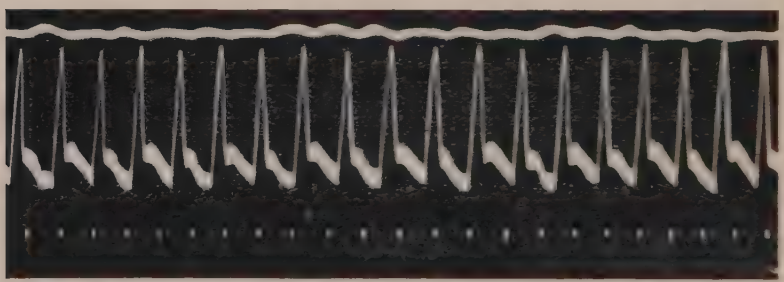

FIG. 99.—Ventricular fibrillation. This record was obtained by experimentally stimulating the ventricles in a cat; a tetanizing electric current applied directly to the exposed heart (from Wenckbach and Winterberg).

phenomenon accompanying fibrillation of the auricles. Its development is of importance in explaining the sudden deaths occurring in coronary artery sclerosis with infarction. As described before, the infarcted area with its necrosing and highly irritable muscle fibres, may become the foci of ventricular extrasystoles; these may develop at such a rate, that paroxysmal tachycardia may ensue, and the transition to fibrillation of the ventricles set in.

PULSUS ALTERNANS

An interesting and rather infrequent exhibition of the pathologic response of the heart muscle is seen in the condition, described for many years as *pulsus alternans*. This condition is usually manifest only by an examination of the hemodynamic factors of the cardiac cycle, as electrocardiographic studies alone may fail to reveal its presence. Radial pulse tracings show an alternation of large and small beats coming through to the wrist. Its discovery is to be regarded with special significance, as it is only seen in cardiovascular systems which have been the site of considerable pathology. Few cases have been found to recover from this condition. Theoretically, pulsus alternans is associated with disturbances in the reparative process following myocardial infarction. No electrocardiographic evidence may be given of the condition, but occasionally the alternation is so well marked, that even the electrodynamic activity of the heart is affected and the QRS complexes may be alternately larger and smaller. There may be no correspondence, however, between the large QRS complexes and the large ventricular contractions; in fact, the reverse may be found, i.e., small QRS complexes with each of the larger radial beats and large QRS complexes with the small radial beats may occur.

PSEUDO-PULSUS ALTERNANS

Rather more frequently than the actual occurrence of pulsus alternans, may arise disturbances of rhythm which superficially resemble it. The palpating finger at the radial pulse may be misled by the finding of alternating large and small beats. The most common condition producing this alternation is the occurrence of extrasystoles developing late in diastole. Such extrasystoles, because they are premature, cause contraction of the left ventricle before it is

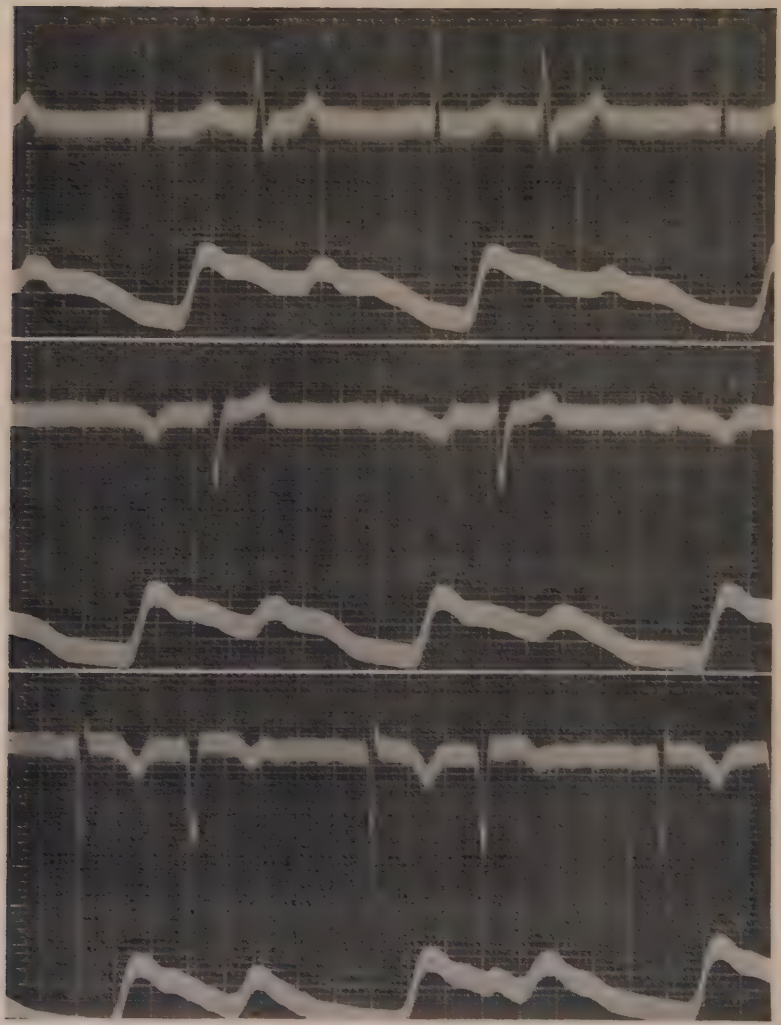

FIG. 100.—Pseudo-pulsus alternans. As the result of an extrasystolic arrhythmia, every other radial pulsation appears to be smaller. Analysis of the polygraphic tracing, however, will show that the smaller beats are somewhat premature and that there is a larger interval between the smaller and larger beat than between the larger and smaller one. The electrocardiographic tracing shows this very clearly.

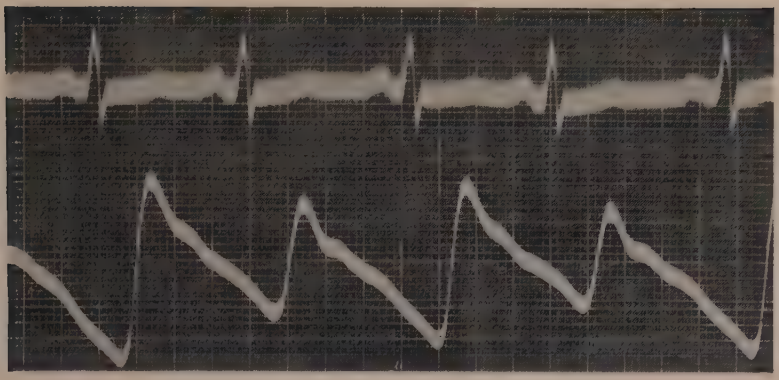

FIG. 101.—Pseudo-pulsus alternans. As a result of an unusual disturbance of the pacemaker every other impulse is released a little prematurely so that the ventricles are not completely filled and a smaller pulsation is felt at the radial pulse. The normal impulse taking a longer time to generate, permits a longer diastolic period and hence a greater volume of blood is expelled on ventricular contraction and a larger pulsation is felt at the wrist. Palpation of the radial artery gives the impression of alternating larger and smaller beats, but measurement of the radial upstroke in the polygraphic tracing will show alternating longer and shorter periods.

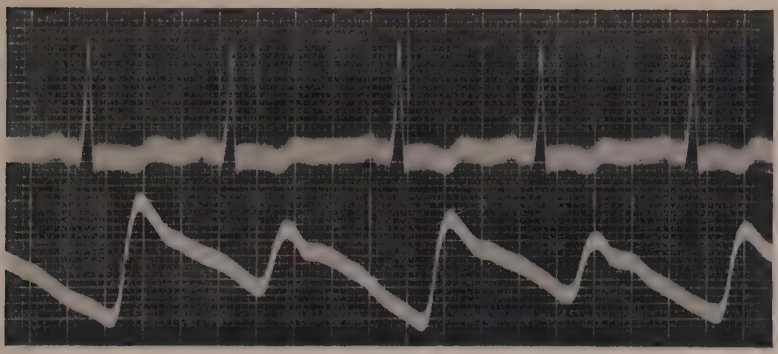

FIG. 102.—Pseudo-pulsus alternans occurring in hypertension. Note here the associated inversion of the T wave in the first lead.

completely filled so that the pulsation at the wrist is smaller than that following a normal beat.

Another form of pseudo-pulsus alternans is seen in certain rare disturbances of the pacemaker mechanism, where there is alternating delay in the release of the impulse. This phe-

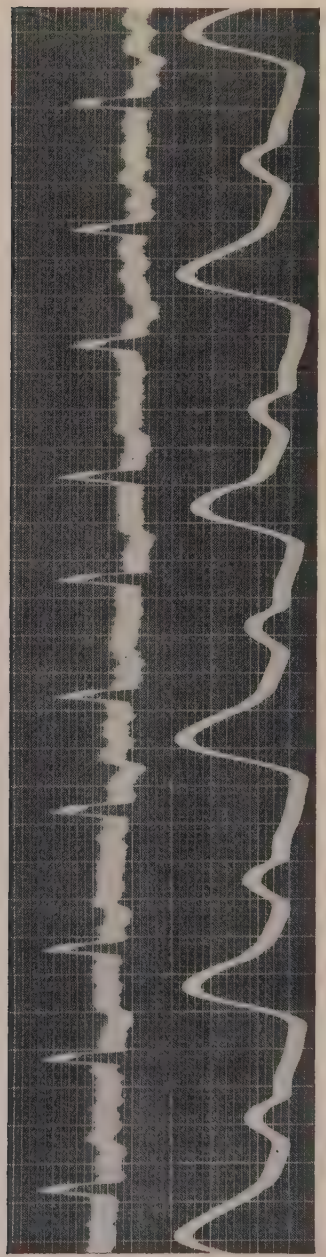

FIG. 103.—Pseudo-pulsus alternans occurring in a case of auricular fibrillation. This condition is extremely rare. Because of some vague disturbance occurring in a physiological block at the atrio-ventricular junction, every other impulse which is permitted to come through occurs at a slightly shorter time interval. The same filling phenomenon of the ventricles takes place as seen in the previous two records. This case is interesting in that a diagnosis of pulsus alternans was made by polygraphic methods without the auricular fibrillation having been disclosed until electrocardiographic studies were made.

nomenon has been previously described in the chapter on diseases of the pacemaker, but it can be pointed out here that the alternating delay in the release of the impulse allows the ventricle at every other beat to become more completely filled. In this way, those contractions of the more nearly filled ventricles are accompanied by a larger radial pulsation, and the impression is thus given to the palpating finger that a condition of pulsus alternans exists.

Of rare occurrence is the development of pseudo-pulsus alternans in auricular fibrillation. This condition has been found only once in more than 5,000 records; it is interesting only in that it deceived the examiner in believing that a true pulsus alternans existed. This condition, however, was readily found to be due to a peculiar rhythm during the course of auricular fibrillation.

The clinical discovery of pulsus alternans by palpation of the radial artery should always be followed by a complete electrocardiographic and polygraphic study of the patient. True pulsus alternans, as its name implies, is a regular rhythm with alternating large and small beats; the regularity of rhythm can not be over emphasized, as the phenomenon of alternation of large and small beats can be produced by any condition which calls forth a regular delay in the contraction of the left ventricle. As noted above, the most common cause of such a delay is due to an extrasystolic arrhythmia. Inasmuch as the diagnosis of true pulsus alternans carries with it such a sinister prognosis, it behooves the clinician to determine the real character of the alternation of beats at the wrist.

CHAPTER VIII

DISTURBANCES OF THE CORONARY ARTERY CIRCULATION

DISTURBANCES OF THE CORONARY ARTERY CIRCULATION

No phase of cardiovascular pathology has received more attention in the past decade than that concerning the degenerative vascular conditions that affect the heart in persons of middle and advancing age. Whether such changes are to be considered merely as evidence of the aging process and hence are normal for a given age period, or whether such changes are actually a well defined clinical entity has been the subject of much discussion and speculation.

Certain it is, however, that fundamental alterations are found in the terminal ventricular complex of the electrocardiographic tracing in various types of cardiac pathology of a serious nature.

Alteration of the terminal ventricular complex or the T wave still constitutes one of the mooted problems as to its true significance. At the same time, however, the discovery of such T wave changes may bring information of the greatest importance to the clinician. This may in certain cases render a diagnosis which would be impossible by any other mode of investigation.

As has been pointed out before, the T wave, like all the other important waves indicated in the electrodynamic activity of the heart, is upright. This holds true in so far as the first and second leads are concerned; the T wave seen in the third lead is subject to many variations and changes in what apparently are normal hearts. At the same time, the T wave of the third lead is more often seen upright than changed in individuals who are to be regarded as normal. For example, in a study made on 500 college students, all of whom had been passed as being physically fit, it was found that 421 had upright T waves in the third lead; 16 had bizarre and irregularly configurated T waves, and 63 had inversions of the T waves of varying degrees.

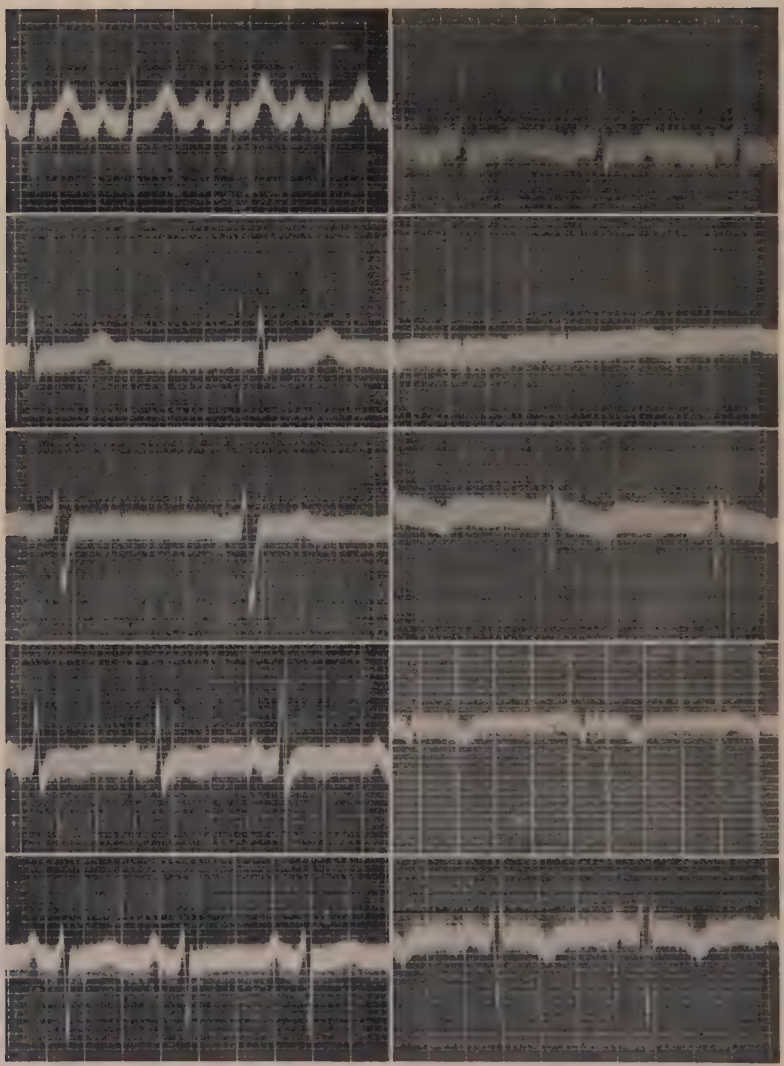

FIG. 104.—A series of tracings made upon normal students, showing the various abnormalities of the T waves found in the third lead.

In our experience, T wave changes in the third lead are only significant in relation to similar T wave changes in Leads I and II.

Very recently a study of T wave inversion of the third lead made by the authors has shown a certain interesting correlation between such T wave changes and habitual con-

FIG. 105.—Alteration of T waves seen in Lead I in a case of mushroom (muscarin) poisoning.

stipation. The significance of this finding may be fruitful of much investigation, and while we have no opportunity to enter into a discussion of the problem in such a volume as

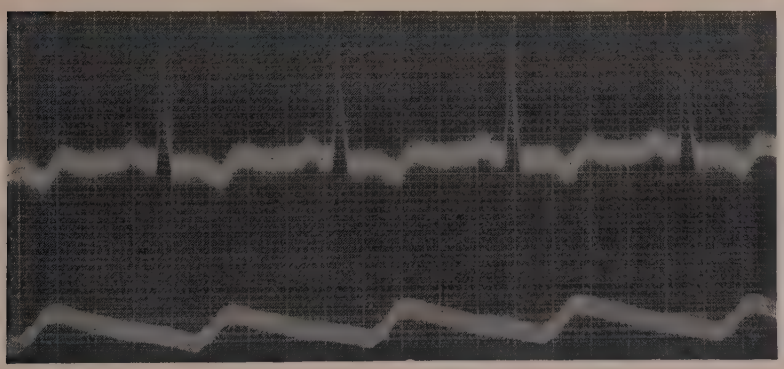

FIG. 106.—Alteration of the T waves in Lead I found in a case of typhoid fever.

this, it is possible to point out that constipation in these cases may have an effect upon the heart like that found in other toxic conditions.

Because of its variability, the T wave in the third lead is generally not considered when found altered alone.

Changes of the T waves in the first and second leads, however, are of far greater import. Such changes have been known to occur in many toxic affections of the myocardium. The condition is readily produced by digitalis when pushed to the point of overdosage and is also seen in muscarin and squill poisoning. Altered T waves are also found during the highly toxic stages of the common acute infectious diseases like diphtheria, pneumonia, and typhoid fever. (See Fig. 74.)

Such T wave alterations are usually of only temporary duration. They may appear and disappear in the space of a few days or weeks. Where extensive myocardial damage has taken place, the condition may persist even for years.

Of far greater interest and of more especial significance, are the alterations found in the T waves of the first and second leads in patients having no history of such toxic affections of the heart. Into this group come what has been recently termed *"The Coronary Artery Syndrome,"* and because of its intimate association with angina pectoris and sudden death the subject has received widespread attention.

Winterberg and Rothberger in 1914, demonstrated in the dog that the main branches of the coronary arteries might be ligated without death occurring. It was shown that if the ligation was done very quickly, the heart would promptly stop beating and death ensued; on the other hand, if the ligation was done slowly over a period of minutes and hours, the heart continued to function. In these dogs certain characteristic changes were given to the T waves in the first and second leads and, because of their constant occurrence in these two leads, the custom has arisen to indicate leads I and II by the phrase—*significant leads.*

Many investigators have subsequently demonstrated the relation between the postmortem findings of coronary artery occlusion and T wave changes in the significant leads. As the degenerative disease process occurring in the coronary arteries differs in no way apparently from that seen in

other vessels of the body, the sequence of events appears to be as follows: the destructive changes occurring in the vessel walls gradually narrow the lumen of the vessel and thereby restrict the passage of blood through it, slowing the circulation. The damaged intimal wall readily becomes the site of thrombus formation which in turn further restricts the passage of the blood stream. This stenosis of the coronary vessels may in itself produce the series of symptoms familiarly known as a *stenocardial syndrome,* with its accompanying anginal picture. Electrocardiographic studies at this time may show little or no especial deviation

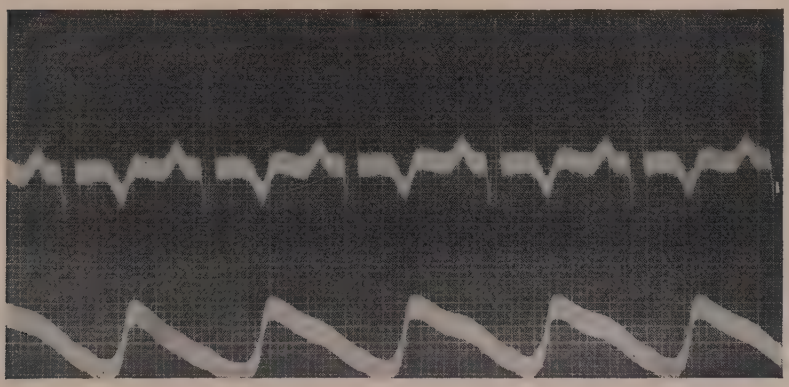

Fig. 107.—Alteration of T wave, bowed type.

from the normal. When, however, the narrowed vessel suddenly becomes occluded by fragmentation and dislodgement of the thrombus, anemic infarction of the heart may result.

If the process occurs very suddenly, and if the vessel occluded is one of the larger divisions of the coronary tree, instantaneous death may result. If, however, the stenosing process has been slow enough to permit establishment of collateral circulation, the occlusion may be accompanied by infarction of a considerable portion of the heart without causing immediate death. The infarcted area passes through all the stages seen elsewhere in the body with this excep-

tion, that because the heart is a hollow organ capable of withstanding a high internal pressure, the infarct may rupture. This is especially true when the infarction is of sufficient size to undergo liquefaction necrosis. The reparative process consists in absorption of the necrotic heart muscle and replacement by fibrous tissue. This scar, being less resistent than the myocardium, stretches particularly if it occurs in the exposed pressure areas of the heart, and in this way partial or complete aneurysmal dilatation of the heart wall occurs.

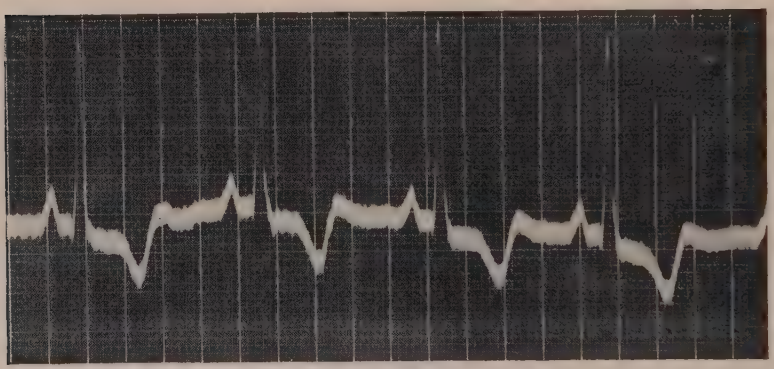

FIG. 108.—T wave alteration of the triangular type.

The electrocardiographic changes found under these pathologic conditions depend in great part upon the location of the damaged myocardium. There apparently exist so-called "silent" areas in the heart, comparable in many respects to those found in the brain; massive areas of the latter may often be damaged or destroyed without producing any clinical signs or symptoms. In the heart, also, large areas of myocardial mischief may exist without instrumental detection. These silent areas occur in those portions of the heart where the conducting system is not well defined. Where, however, the infarct involves or is adjacent to the specialized pathways of impulse conduction, remarkable alterations in the electrocardiographic tracings are found.

The most common of these are the inversions of the T waves in the significant leads; these inversions may assume three well recognized forms: the bowed type, the sharp triangular inversions, and the diphasic form. (Figs. 107, 108, 109.)

The attempt to localize the lesion through the discovery of these various types of T wave alterations has been the subject of much recent study. The problem, however, still lies within the realm of disputed points in electrocardiography. In general, however, it can be said that alteration of

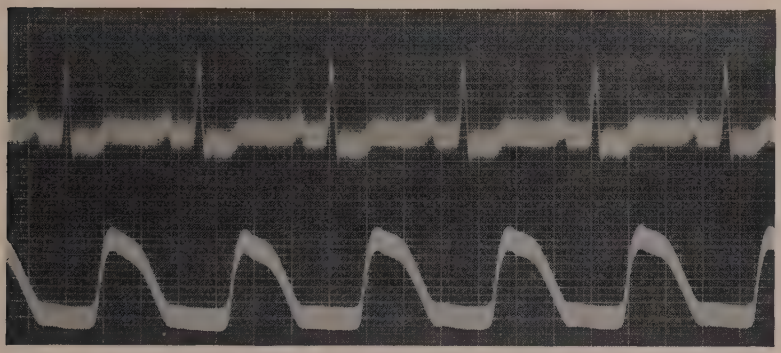

Fig. 109.—T wave alteration of the diphasic type.

the T waves in any of the forms described above is suggestive of serious myocardial damage, and can be regarded with sinister significance so far as prognosis is concerned. The degenerative changes leading to such myocardial mischief are present throughout all the vessels of the heart in the same more or less severe degree, so that having recovered from one infarction, the heart may undergo another or even a third.

At the postmortem table such hearts may show two or three or even four old scarred areas indicating the site of previous infarctions. The general story, however, of such cases is that the degenerative process is continuous, and recovery from one attack is not a guarantee against

subsequent ones. Each attack usually involves a more extensive area and the periods between attacks grow shorter. The prognosis of this condition is to be extremely guarded, and the finding of these T wave alterations have within recent times carried with them a forecast of death within a year or eighteen months. In certain rare instances, patients may live for three or four or even five years after the first discovery of this electrodynamic change. The oldest case in our experience lived for four years and eight months and then died the usual sudden death.

The alteration in the T waves in these degenerative diseases is now regarded as evidence of disturbed coronary circulation. It is better, however, to conceive these changes as being more due to the end results of the altered circulation rather than to the circulatory deficiency itself. As mentioned before, the circulatory deficiency may present the alarming picture seen in the stenocardial syndrome with the characteristic pain, apprehension, and collapse. Electrocardiographic studies in these cases may fail to reveal any T wave change because the coronary stenosis, while functionally of sufficient degree to cause the anginal picture, is not yet severe enough to produce pathologic changes in the heart muscle. Where, however, the stenocardia is due to thrombus formation, the pathology already described may readily occur and give rise to infarctions which destroy the integrity of the myocardium. If the destruction occurs along a pathway of conduction, characteristic electrodynamic alterations will be manifest.

Other evidences of serious myocardial involvement may be indicated through the various types of conduction disturbances found in this condition. Such conduction disturbances depend almost entirely upon the specific anatomic location of the injured muscle. The most common are complete heart block, bundle branch block, and Purkinje fiber block. (See Figures 86, 90, 91, 92.)

Another common manifestation of a disturbed coronary

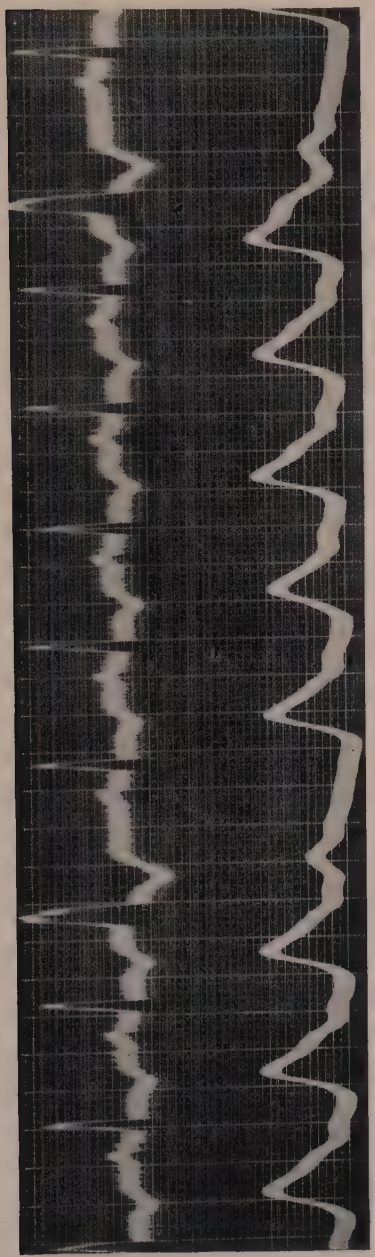

FIG. 110.—Coronary infarction with extrasystoles.

circulation is the development of an extrasystolic arrhythmia. Alterations and restrictions in the blood supply to the ventricular myocardium may result in certain portions of the muscle becoming hyperirritable and, therefore, developing their own stimulus for contraction. (See Chapter IV.) This is especially true when areas of infarction develop; a necrosing myocardial fiber may be the source of many extrasystoles. It has generally been regarded that an extrasystolic arrhythmia developing in the course of a thrombotic attack is of an especially serious significance. Where the extrasystoles are very infrequent, the condition is not so important as when they occur more often. Where they develop very rapidly and produce a sequential rhythm of 10 to 20 beats at a time, the danger of ventricular fibrillation and death is imminent. (See Figs. 57 and 99.)

CHAPTER IX

ELECTROCARDIOGRAPHIC CHANGES IN ACUTE INFECTIOUS DISEASES

ELECTROCARDIOGRAPHIC CHANGES IN ACUTE INFECTIOUS DISEASES

The discovery of electrodynamic changes occurring during the course of the acute infectious diseases has within recent times added considerable value to electrocardiographic interpretations. This field of study has unfortunately not received sufficient attention among clinicians

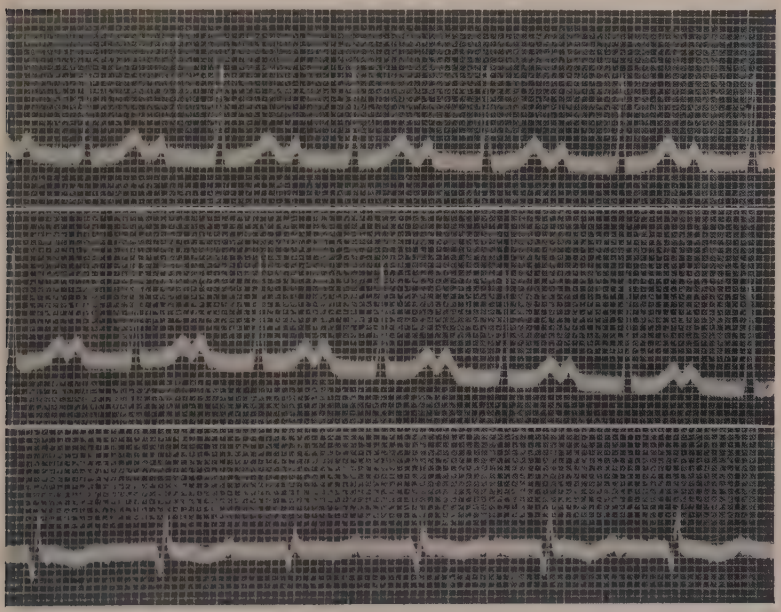

Fig. 111.—Prolongation of P-R interval during attack of influenza. (P-R interval measures 0.34 seconds.)

and those interested in cardiovascular pathology. The electrocardiograph becomes an instrument of considerable prognostic importance when used intelligently as a control in convalescence from even the most simple infectious diseases.

The electrodynamic changes which occur in the heart during an attack of acute rheumatic fever have been known

for many years; similar changes have been found to occur in diphtheria, typhoid fever, pneumonia, and even in influenza. These changes are usually found in the conducting system and are especially common at the auriculo-ventricular junction. These are expressed by a lengthening of the P-R interval in the electrocardiographic tracing. Prolongations of the P-R interval to 0.24, and as high as 0.32 are not uncommon. In severe cases of toxic infection, second degree heart block may occur, and very rarely complete heart block.

Pathologically, there is considerable evidence to show that the delay in the conducting system is probably due to a toxic edematous condition of the bundle fibres, and its occurrence is usually associated with widespread myocardial mischief.

From a clinical point of view, a patient may have run a typical course of acute rheumatic fever, diphtheria, or influenza and to have made a complete recovery. Convalescence is ordinarily left to the whim and feelings of the patient or his family, no attention being paid to residual disease which may be left in the heart and vascular system. The patient may undertake work entailing effort to such a degree that the heart muscle may become irremediably overstrained and permanent damage to the muscular elements of the cardiovascular system may ensue.

Electrocardiographic study of these conditions has shown that conduction disturbances may persist for many days and even weeks after the patient has been pronounced clinically well. Heart strain under these conditions can well produce changes of a permanent character. Just so long as conduction disturbances are present, it can be assumed that the heart tissues are undergoing a definite toxic reaction to which the addition of heart strain may make recovery doubtful.

We have found that there is no other method of examining such a patient to determine the duration of convales-

cence. The usual signs, like persistent temperature and leucocytosis may not reveal themselves. Electrocardiographic studies, however, may show the condition very clearly and simply. (See Figs. 79, 80, and 81.)

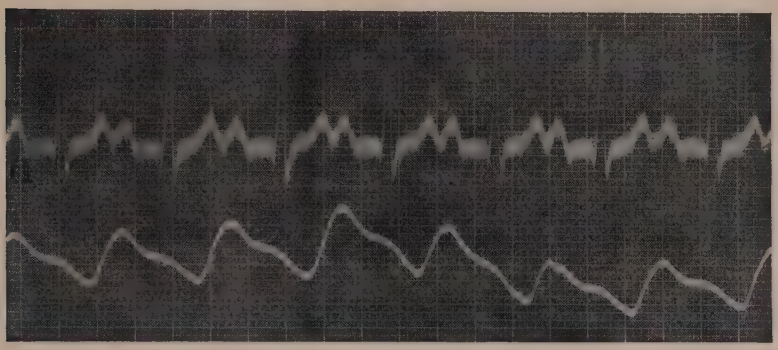

Fig. 112.—Same case after clinical recovery. Note that P-R interval is still delayed to 0.28 seconds.

We believe that the routine electrocardiographic study of all patients convalescing from any of the acute infectious diseases would result profitably to the patient and should be used as a guide in the resumption of his usual activities. *Much subsequent heart disease might be prevented if such prophylactic measures were generally employed.*

CHAPTER X

VALVULAR HEART DISEASE IN RELATION TO ELECTROCARDIOGRAPHY

VALVULAR HEART DISEASE IN RELATION TO ELECTROCARDIOGRAPHY

The fundamental conception of cardiovascular disease has passed through four successive phases of medical philosophy. The very early clinicians following in the footsteps of Harvey and his contemporaries viewed the various maladies of the heart as manifestations of anatomical change alone. With the discovery that a defective valve mechanism gave rise to certain sounds or murmurs, the attention of that medical period was focused upon this phenomenon, practically to complete exclusion of the former viewpoint. The diagnosis of heart murmurs became paramount; valvular disease of the heart was the predominant factor considered in relation to the presence or absence of cardiovascular pathology. This viewpoint remained unchallenged until the histologic structure of the heart became more thoroughly known. With the development of our knowledge of the specific conducting system and the pacemaker mechanism, the rôle of the irregularities of the heart beat assumed prime importance. The researches of Mackenzie, Vaquez, Winterberg, Wenckebach, and Lewis, served to complete the conception of anatomical and functional disturbances of the heart. The past generation has witnessed intense study of the various irregularities of the cardiovascular system made possible by the utilization of special instruments like the polygraph, electrocardiograph, and X-ray. The fourth and present phase, taking full cognizance of previous achievements made in the study of heart disease has given preëminence to etiology.

It is believed to-day that all of the various changes occurring in the different structures of the cardiovascular system are the result of certain well defined pathological processes. These processes appear to divide themselves into definite age groups; in youth the infectious conditions are

preëminent; rheumatic fever, diphtheria, and scarlet fever may leave irreparably damaged hearts which subsequently pass through the various valvular and conduction disturbances. By far the most important etiologic factor of heart disease in individuals under the age of thirty is due to rheumatism. With middle and advancing age periods the degenerative changes accompanying the aging process loom as the principal factors in heart disease. In addition to the above must be mentioned syphilis which will affect the heart through the individual's entire life period, because of its decided predilection for the cardiovascular system.

From the very beginning of the clinical application of the electrocardiograph, the attempt has been made to correlate valvular changes with electrodynamic phenomena. The enthusiasm accompanying the application of any new technical method is frequently followed by a pessimistic reaction. This by no means detracts from the usefulness of this instrument, since its limitations are now fully recognized.

The question is frequently asked whether the electrocardiogram is an aid in the diagnosis of valvular heart disease; this question calls to attention the undue emphasis which is still placed upon valvular defects alone, and many clinicians upon learning that this method may render no additional information turn away with the thought that the electrocardiograph is of little value.

Not until the cardiodynamic activity has been altered by well marked anatomic change in the heart structures will there appear any deviation from the normal in the electrocardiographic record. The most frequent changes occurring as a result of valve disfunction are those resulting in dilatation or hypertrophy of the heart chambers. Undoubtedly, the most common alterations from the normal are found in changes of the left ventricle; such changes may be the result of aortic or mitral disease. This left ventricular enlargement is associated with a rotation of the heart

toward the left and is accompanied by a corresponding deviation in the electrical axis of the heart. Electrocardiographically this will be indicated by noting the QRS complexes in the first and third leads; the complex will be upward in Lead I and downward in Lead III.

While left axial deviation is commonly found in aortic and mitral disease, other conditions which cause enlargement of the left ventricle may give the same findings, even in the absence of valve deficiencies. Such conditions as hypertension, aortitis, aneurysm of the aorta, mediastinal tumors displacing the heart to the left, pericardial and right sided pleural effusions, all tend to displace the electric axis toward the left and present in the electrocardiogram alterations of similar nature.

On the other hand, stenosis of the mitral valve may be associated with right ventricular hypertrophy because of left auricular interference. This change in the right ventricle tends to rotate the entire heart toward the right and a right axial deviation results. This is indicated electrocardiographically by the QRS complex being downward in the first lead and upward in the third. Such right axial deviation of the heart, however, is not pathognomonic of mitral stenosis alone, since a similar deviation may arise in any event which displaces the heart toward the right. These conditions are very similar to those mentioned in left axial deviation, the only difference being that of direction. In general, therefore, it may be said that right or left axial deviation of the heart is only of importance in relation to established clinical findings.

In valvular disease alone the electrocardiograph renders practically no additional information. Certain changes are, however, observed in some valvular defects, the most striking of which are the changes in the P wave occurring in mitral stenosis. Because of the pathology causing hypertrophy of the left auricle, either large or split P waves are found; in addition, the conduction time from the auricles

to the ventricles may be increased so that the P-R interval is prolonged.

Since, however, the etiological factor in heart disease is assuming more and more importance, electrocardiographic examination in valvular disease may aid in determining the extent or presence of other lesions within the heart. The functional ability of the heart muscle to overcome the obstacles of valve deficiency is of far greater importance than the actual type and extent of valve change; a sound heart muscle may be very little affected by such valvular defects. When alterations are found in the ventricular complexes in association with valve changes the prognosis is far more serious than when normal tracings are obtained.

CONGENITAL HEART DISEASE

The congenital malformations and anomalies of the heart may sometimes give characteristic electrocardiographic changes if there has been a disturbance in the electrical axis of the heart. This is especially true in regard to *dextrocardia.* In this condition, all of the waves in the first lead are usually inverted but there is no change in the configuration of the waves in Leads II and III.

The electrocardiograph may be of considerable clinical value in distinguishing between true congenital dextrocardia and one acquired as a result of conditions arising in the lungs or thoracic cavity. Pleurisy with effusion, empyema, and diseases of the mediastinum, especially on the left side, may push the heart over into the right chest and thereby change the electrical axis of the heart. Such cases, when studied electrocardiographically show a typical right axial deviation but no inversion of the P waves or T waves in the first lead.

Where some doubt may exist as to whether the dextrocardia is acquired or congenital, because of the difficulty in interpreting the P and T waves in the first lead, the elec-

trodes leading from the two arms may be reversed according to the method described by Rothberger; the electrode from the right arm is placed on the left and that from the left

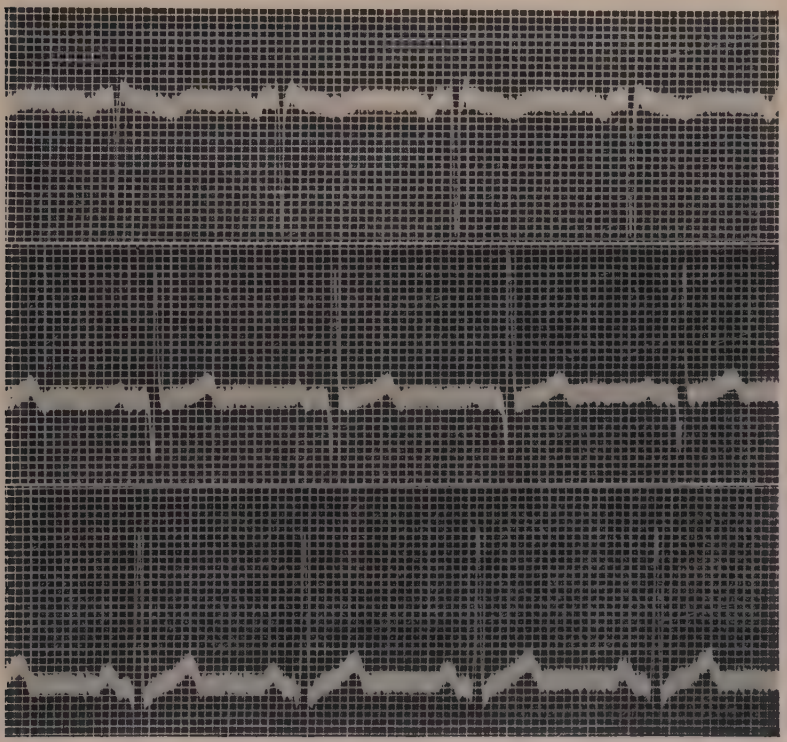

Fig. 113.—Congenital dextrocardia. Note inversion of all waves in Lead I.

on the right. In congenital dextrocardia, the first lead taken in this manner will show all of the waves upright where they have been inverted before, while in acquired dextrocardia they will remain unchanged.

CHAPTER XI

ANALYSIS OF ELECTROCARDIOGRAPHIC TRACINGS

SCHEMA FOR ELECTROCARDIOGRAPHIC READING

1. RHYTHM. Note if it is irregular or regular, presence or absence of sinus arrhythmia. (Remember that a sinus arrhythmia, particularly in childhood, is a diminuendo and crescendo effect, and the rate of the beat follows a very definite schema).

   Do not confuse it with irregularly spaced intervals.

   If the rate is irregular, note whether it is due to one of the four following causes:—

   a. Auricular fibrillation.
   b. Auricular flutter.
   c. Extrasystoles.
   d. Heart block.

2. P WAVE: Observe the following:—
   a. Absence.
   b. Shape.
   c. Inversion.
   d. Length of P-R interval and its constancy.

3. QRS COMPLEX:
   a. Preponderance.
   b. Width of transmission time.
   c. Notching or splitting.
   d. Arborization or feathering.
   e. Respiratory variant, particularly in third lead, and also note alternation in height.

4. T WAVE:—
   a. Presence or absence.
   b. Position—upright or down.
   c. Irregularities in form.
   d. Large forms.

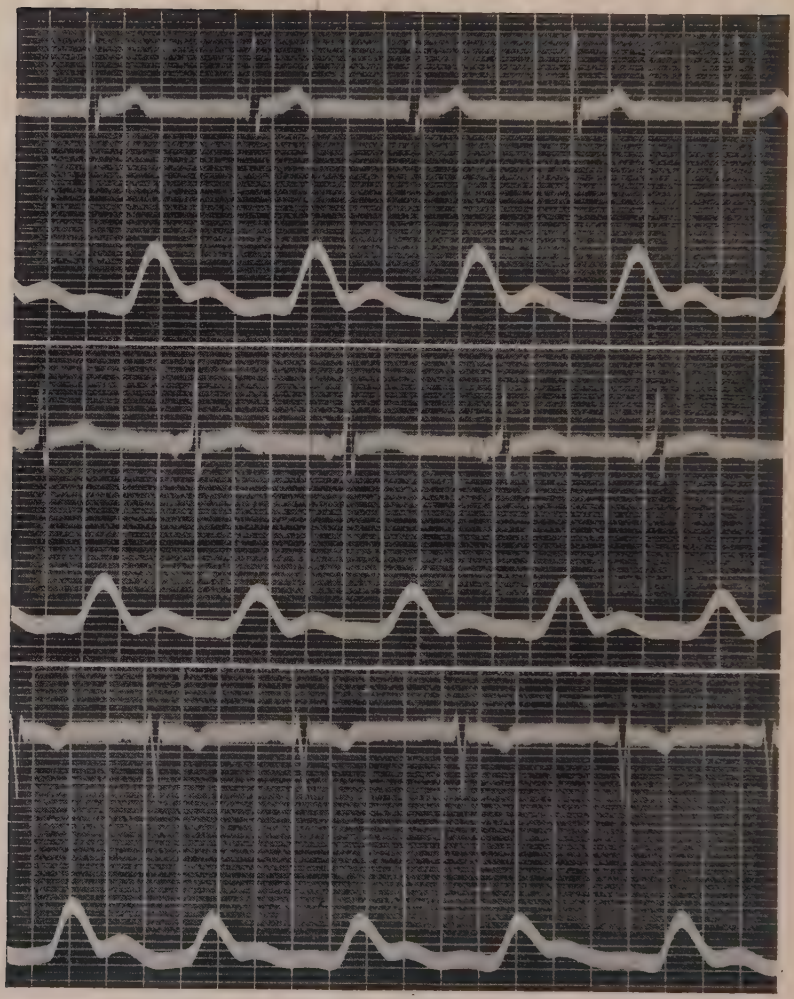

FIG. 114.—Normal record showing maximum number of variations. Man, aged 42; no symptoms, negative physical examination. Variations: P waves absent in Lead I; inverted in Lead II; upright but split in Lead III. QRS complex upright in Lead I but downward in Lead III suggesting a left axial deviation; normal for this age and type. T waves inverted in Lead III.

DETAILED ANALYSIS OF ELECTROCARDIOGRAPHIC TRACINGS

SECTION I. CHANGES OF THE P WAVE

1. THE P WAVE IS WANTING OR ABSENT IN

a. In Lead III it is frequently absent normally.
b. Auricular fibrillation.
c. In complete heart block.
d. In auriculo-ventricular nodal extrasystoles.
e. Escaped auriculo-ventricular nodal beats.

2. P WAVE NEGATIVE OR INVERTED IN

a. Dextrocardia in Lead I.
b. In auricular extrasystoles arising near the auriculo-ventricular node.
c. In extrasystoles arising near the atrio-ventricular border.

3. P WAVE UNUSUALLY HIGH IN

a. Hypertrophy of auricles as in mitral stenosis.
b. In irritation of the accelerator nerves.

4. P WAVE SPLIT IN

a. Sometimes in normal tracings.
b. Hypertrophy of auricles as in mitral stenosis.

5. P WAVE IS BLENDED OR COALESCES WITH THE T WAVE PARTLY OR COMPLETELY IN

a. Second or third degree heart block.
b. In ordinary sinus tachycardia.
c. Paroxysmal tachycardia.
d. In very premature auricular extrasystoles.
e. In very premature auriculo-ventricular extrasystoles arising near the border,—the inverted P wave coalesces with the T wave. (Note that it is possible that the T wave may be inverted and coalesce with an upright P wave, or the T wave may be upright and may coalesce with an in-

verted P wave, or both the T or P waves may be inverted.)

6. THE P WAVE FOLLOWS THE QRS COMPLEX IN

a. Escaped beats.

b. In some auriculo-ventricular extrasystoles with dissociation of auricles and ventricles.

7. THE P WAVE OCCURS MORE FREQUENTLY THAN THE QRS AND T COMPLEXES IN

a. Auricular flutter.

b. In partial or second degree heart block.

c. In complete heart block.

SECTION II. CHANGES THAT MAY OCCUR IN THE P-R INTERVAL

1. SHORTENING

a. Auricular extrasystoles.

b. In escaped beats with dissociation of auricles and ventricles.

2. LENGTHENING

a. Conduction disturbances.

b. Mitral stenosis.

SECTION III. CHANGES IN INITIAL DEFLECTION

1. Q WAVE IS ABSENT

a. Normally absent very often in leads I and II.

2. Q WAVE MAY BE VERY DEEP

a. Suggests degeneration of the septum. Ex.-Infarct, gumma, tubercle.

3. S WAVE MAY BE ABSENT

a. Normally in any one lead.

b. Mechanically in loose string.

4. S WAVE UNUSUALLY DEEP

a. In disease of the anterior papillary muscle of left ventricle?

5. QRS COMPLEX ABSENT

a. Partial heart block.
b. Complete heart block.
c. Auricular extrasystoles with block.
d. Auricular flutter.

6. QRS INVERSION

A. Lead I.
a. Dextrocardia.
b. Right axial deviation.
c. Left bundle branch block.
d. Right ventricular extrasystoles.

B. Leads II and III
a. Left ventricular extrasystoles.
b. Left axial deviation.
c. Right bundle branch block.

7. INITIAL DEFLECTION IS SPLIT

A. Simple splitting.
a. In Lead III normally.
b. In all leads in various forms of heart disease.
c. In supraventricular extrasystoles.

B. Multiple splitting.
a. Ventricular extrasystoles.
b. Bundle branch block.
c. Myodegeneration.

8. INITIAL DEFLECTION WIDENED

a. Bundle branch block.
b. Extrasystoles.
c. Myodegeneration.

SECTION IV. CHANGES IN THE T WAVE

1. T WAVE WANTING

a. Normally in Lead III.
b. In Leads I and II in general weakening of the myocardium.

2. T WAVE IS INVERTED

a. Normally in Lead III.
b. In Leads I and II in coronary occlusion.
c. In digitalization.
d. In Lead I in dextrocardia.

3. T WAVE ESPECIALLY HIGH

a. Irritation of accelerator nerves.
b. In Lead II in hyperthyroidism.

SECTION V. CHANGES IN THE T-P INTERVAL

1. SHORTENING

a. Tachycardia.

2. LENGTHENING

a. Sinus arrhythmia, on deep inspiration.
b. Blocked supraventricular extrasystoles.
c. Sino-auricular block or nodal depression.
d. Complete heart block with slow node of Tawara rhythm.

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